A RANDOMIZED STUDY OF PREVALENCE OF PRIMARY OPEN ANGLE GLAUCOMA IN PATIENTS AGED 40 AND ABOVE ATTENDING OPHTHALMOLOGY OUTPATIENT DEPARTMENT IN A TERTIARY HOSPITAL

DISSERTATION SUBMITTED TO

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M.S. DEGREE EXAMINATION OF

BRANCH III OPHTHALMOLOGY

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CERTIFICATE

This is to certify that this dissertation entitled **A randomized study of prevalence of primary open angle glaucoma in patients aged 40 and above attending Ophthalmology outpatient department in a tertiary hospital** submitted by **Dr.P.Bavaanni preetti** is a bonafide research work carried out by her under our direct supervision and guidance. This dissertation is submitted to The Tamil Nadu Dr. MGR Medical University, Chennai towards the partial fulfillment of the requirements for the award of M.S Degree (Branch III) in Ophthalmology.

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ABSTRACT

A randomized study of prevalence of primary open angle glaucoma in patients aged 40 and above attending Ophthalmology outpatient department in a tertiary hospital

Aims and Objectives

To study the prevalence of primary open angle glaucoma in patients aged 40 and above attending Ophthalmology outpatient department in Tirunelveli Medical College Hospital and to evaluate their awareness and knowledge about glaucoma .

Materials and methods

One hundred patients aged 40 years and above attending Ophthalmology outpatient department in Tirunelveli Medical College Hospital from January 2012 to September 2013 underwent complete ophthalmic examination.

Results

The overall prevalence of primary open angle glaucoma was 5% and the prevalence of glaucoma suspect was 1% . Diabetes and hypertension did not have any significant etiological association with primary open angle glaucoma. The mean IOP in the right eye was 14.22 $\pm$ 3.37 mm Hg with applanation tonometer, 14.50 $\pm$3.38 mm Hg with non contact tonometer and 15.41$\pm$ 3.19 mm Hg with Schiotz tonometer. The mean IOP in the left eye was 13.87 $\pm$3.35 mm Hg with applanation tonometer, 14.45 $\pm$3.56 mm Hg with non contact

tonometer and 15.22± 3.18 mm Hg with Schiotz. IOP found with applanation tonometer was comparable with non contact tonometer in the right eye (p 0.133) but there was a significant difference between them in the left eye (p 0.001).There was a significant difference between applanation tonometer and Schiotz in both eyes ($p<0.001$). Visual field changes had good correlation with the optic disc changes. Cup disc ratio and peripapillary atrophy had good correlation with primary open angle glaucoma. The mean cup disc ratio in the right eye was 0.31 in non glaucomatous and 0.64 in the primary open angle glaucoma patients. The mean cup disc ratio in the left eye was 0.32 in non glaucomatous patients and 0.62 in the primary open angle glaucoma patients. 60% of the primary open angle glaucoma patients were diagnosed newly. Glaucoma awareness was extremely low (8%) and knowledge was 0%. There was no significant association between literacy and glaucoma awareness. Previous eye check up and eye camps were not effective in creating awareness in the patients.

Conclusion

The overall prevalence of primary open angle glaucoma was 5% and the prevalence of glaucoma suspect was 1%. Fundus examination to rule out glaucoma is mandatory in all patients above 40 years of age. Glaucoma awareness (8%) and knowledge (0%) was extremely low considering the magnitude of blindness due to glaucoma. Steps to promote awareness among the public and health care personnel must be initiated.

LIST OF ABBREVIATIONS USED

POAG	primary open angle glaucoma
IOP	intraocular pressure
BP	blood pressure
RNFL	retinal nerve fibre layer
ONH	optic nerve head
GAT	Goldmann applanation tonometer
NCT	non- contact tonometer
HFA	Humphrey Field Analyzer
TOP	tendency- oriented perimeter
NTG	normal tension glaucoma
e.g	example
WGA	World Glaucoma Association
WGPA	World Glaucoma Patient Association
APEDS	Andhra Pradesh Eye Disease Study
ACES	Aravind Comprehensive Eye Survey
CGS	Chennai Glaucoma Study
VES	Vellore Eye study
WBGS	West Bengal Glaucoma Study
CDR	cup disc ratio
PPA	peripapillary atrophy

ABBREVIATIONS USED IN RESULTS

RE	right eye
LE	left eye
M	male
F	female
S	Schiotz

INTRODUCTION

Glaucoma is considered the “silent killer of sight”. Until the advanced stage, it is asymptomatic. Glaucoma is an irreversible condition hence early detection and treatment is essential for the control of blindness due to glaucoma.

The case detection rates must be increased by increasing the awareness about glaucoma, thereby reducing blindness due to glaucoma. Early detection of glaucoma through ‘opportunistic case detection’ by performing a comprehensive eye examination at all levels and all available instances, and appropriate referral or initiating treatment as early as possible will help to improve the patient’s quality of life.

EPIDEMIOLOGY

Prevalence

The prevalence of primary open angle glaucoma (POAG) varies between various ethnic groups and races. The prevalence is lower in Whites (1.3%) when compared to the blacks (4.7%). Singapore Chinese 2.4%, Japanese 2.6%, Indians 1.7% but Alaskan Inuits (0.1%) and Mongols (0.5%) have lower rates of prevalence. In Ghana,[1] the prevalence is 8% in persons aged above 40.

Age has more significant influence on POAG than race or ethnicity. Before 40 years of age, POAG is uncommon. Prevalence was seen to increase from 0.6% (40-49 years) to 7.33% in those above 80 years.

Incidence

The 4 year incidence was found to be 2.2%[2]. It was found to be age dependent – 1.2% in 40 to 49 years to 4.2 % in those aged above 70 years. Studies from Framington, Rotterrdom, Australia and Minnesota have reported similar age dependent incidence rate of POAG.

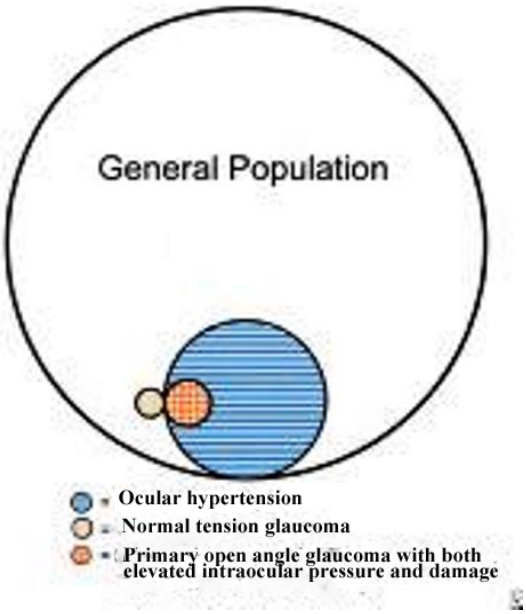
NATURAL HISTORY OF POAG

It can be divided into 3 phases– latency phase, detectable preclinical phase and clinical phase.

The latency phase begins with the onset of glaucomatous optic nerve damage and extends up to the detection threshold.

The detectable preclinical phase begins from the lengthy asymptomatic phase during which glaucoma is detectable until glaucomatous optic nerve damage that leads to symptoms. The detection threshold for glaucoma is the point at which glaucomatous optic nerve damage can be detected accurately by diagnostic testing.

The clinical phase begins with the onset of symptoms.



Using a “ rule of tens ”, we can roughly approximate the distribution of a White or Black population into categories of POAG. For every 1000 persons aged 40 years and older, 100 are suspected of having POAG on the basis of field, disc, IOP findings or dense risk factors; 10 have POAG and 1 will be blind due to POAG.

CLINICAL RISK FACTORS

Identification of risk factors may lead to early intervention and disease prevention strategies. Characteristics that affect risk of glaucoma may also predict the role of progression of disease.

GENERAL

1. Age

The prevalence rates doubles for each decade over 40 years (relative risk of 2 for each decade) and is about 10 fold high in above 80 age group compared to the 40 to 49 age group. The proportion of individual with optic nerve damage and visual loss increases from 1% in persons less than 40 years to 3 to 8 times higher in individuals above 70 years of age. [3]

2. Race

The prevalence is highest in the Blacks[4], intermediate in Whites, Hispanics and South Asian population and lowest in North Asian population.

3. Family history

Increased risk of POAG in having a first degree relative with glaucoma.[5] Around 10–20 % of glaucoma patients have a family history. The association is stronger when the affected relative is sibling rather than a parent or child.

4. Diabetes

Diabetes commonly affects the vascular tissues. But the neuronal and glial tissues in the retina are also compromised leading to apoptosis of the retinal ganglion cells. The neurons and the glia that are already under a compromised state due to diabetes becomes easily susceptible to the added on stress such as elevated intraocular pressure caused by POAG.[6]

When compared to the general population, diabetes have a higher prevalence of POAG and ocular hypertension.

The prevalence of diabetes or a positive glucose tolerance test has also been shown to be higher in patients with POAG and steroid responders.

Diabetes also appears to influence the nature of visual field loss in patients with POAG, with a prevalence of inferior field loss of 64.4% versus 36.4% in diabetics versus non-diabetics, respectively, and a 32% prevalence of diabetes among POAG patients with primarily inferior loss, compared to 13% in those without such a defect.[7]

5. Systemic hypertension

Individuals with systolic blood pressure above a threshold of 130 mmHg had a higher prevalence of open angle glaucoma compared with those with lower systolic blood pressure. Nocturnal arterial hypotension is more common in normal tension glaucoma than in primary open angle glaucoma with elevated intraocular pressure (IOP).[8]

The optic disc capillary circulation may be more precarious as blood pressure (BP) increase and that resultant impaired perfusion of the optic disc may play a contributory role in producing glaucoma.

6. Migraine and Vasospasm

Migraine which may be associated with transient alterations of ocular blood flow and peripheral vasospasm have been suggested as risk factors for open angle glaucoma. This is more closely associated with normotensive glaucoma.[9]

OCULAR

1.Intraocular pressure

It is both a risk factor and cause of glaucoma. Reducing IOP by an average of 23% decreased the incidence of POAG by 60%.[10] Greater pressure lowering results in less progression and stable visual fields.[11]

2.Optic nerve head and peripapillary features

Disc haemorrhages have an elevated risk for progressive visual field loss. It is associated with normal tension glaucoma.[12]

Peripapillary atrophy correlates with the presence of glaucoma but not specific for it. Peripapillary atrophy may worsen along with glaucoma progression. Zone beta atrophy is more common with POAG.

3. Myopia

Myopia is a risk factor for glaucoma with higher prevalence in myopes exceeding 6 dioptres.[13]

4.Others

Thin corneal thickness[14] and exfoliation syndrome are more associated with increased risk for progression.

CLASSIFICATION OF GLAUCOMAS

Glaucomas is classified[15] based on :

The etiology (i.e the underlying pathology that causes alteration of aqueous humour dynamics)

-Primary (no obvious systemic or other ocular disorders)

-Secondary (associated with ocular or systemic abnormalities)

The mechanism (i.e increase in IOP caused by alteration in the anterior chamber angle)

-Open angle glaucoma

-Angle closure glaucoma

CLASSIFICATION OF GLAUCOMAS BASED ON MECHANISM

OPEN ANGLE GLAUCOMA

1) Pretrabecular (membrane overgrowth)

a) Fibrovascular membrane (neovascular glaucoma)

b) Endothelial layer with descemet membrane like membrane

- Iridocorneal endothelial syndrome

- Posterior polymorphous dystrophy

- Penetrating and non penetrating trauma

c) Epithelial downgrowth

d) Fibrous ingrowth

e) Inflammatory membrane

- Fuch's heterochromic iridocylitis

- Luetic interstitial keratitis

2) Trabecular form

a) Idiopathic

- Chronic open angle glaucomas

- Steroid-induced glaucomas

b) Clogging of the trabecular meshwork

- Red blood cells

Hemorrhagic glaucoma

Ghost cell glaucoma

-Macrophages

Hemolytic glaucoma

Phacolytic glaucoma

Melanomalytic glaucoma

- Neoplastic cells

Malignant tumours

Neurofibromatosis

Nevus of Ota

Juvenile xanthogranuloma

-Pigment particles

Pigmentary glaucoma

Exfoliation Syndrome

Uveitis

Malignant melanoma

-Protein

Uveitis

Lens-induced glaucoma

Viscoelastic agents / postoperative

Silicone oil

Alpha-chymotrypsin induced glaucoma

Vitreous

c) Alterations in the trabecular meshwork

- Edema

Uveitis (Trabeculitis)

Scleritis and episcleritis

Alkali burns

- Trauma (angle recession)

- Intraocular foreign bodies (Hemosiderosis, Chalcosis)

3) Posttrabecular form

a) Obstruction of Schlemm canal

- Collapse of canal

- Clogging of canal (e.g. Sickled RBCs)

b) Elevated episcleral venous pressure

- Carotid -cavernous fistula

- Cavernous sinus thrombosis

- Retrobulbar tumours

- Thyrotropic exophthalmos

- Superior vena cava obstruction

- Mediastinal tumours

- Sturge-Weber Syndrome

- Episcleral venous pressure elevation

ANGLE CLOSURE GLAUCOMA

1) Anterior forms ("Pulling" mechanism)

a) Contracture of membranes

Neovascular glaucoma

Iridocorneal endothelial syndrome

Posterior polymorphous dystrophy

Penetrating and non penetrating trauma

b) Contracture of inflammatory precipitates

2) Posterior ("Pushing" mechanism)

a) With pupillary block

- Pupillary block glaucoma

- Lens induced mechanism

Intumescent lens

Subluxation of lens

Mobile lens Syndrome

-Posterior synechiae

Iris-vitreous block in aphakia

Iris-intraocular lens block in pseudophakia

Uveitis with posterior synechiae

b) Without pupillary block

- Plateau Iris Syndrome

- Ciliary block (Malignant) glaucoma

- Lens induced mechanisms

Intumescent lens

Subluxation of lens

Mobile lens syndrome

- Following lens extraction (forward vitreous shift)

- Following scleral buckling

- Following pan retinal photocoagulation

- Central retinal vein occlusion

- Intraocular tumours

    Malignant Melanoma

    Retinoblastoma

- Cysts of the iris and ciliary body

- Retrolenticular tissue contracture

- Retinopathy of prematurity

- Persistent hyperplastic primary vitreous

DEVELOPMENTAL ANOMALIES OF THE ANTERIOR CHAMBER ANGLE

1) High insertion of anterior uvea

    Congenital (infantile) glaucoma

    Juvenile glaucoma

2) Incomplete development of trabecular meshwork/ Schlemm canal

    Axenfeld -Rieger syndrome

    Peter's anomaly

    Glaucomas associated with other developmental anomalies

3) Iridocorneal adhesions

    Broad strands (Axenfeld- Rieger Syndrome)

    Fine strands which contract to close angle (aniridia)

PRIMARY OPEN ANGLE GLAUCOMA

It is also known as chronic open angle glaucoma and chronic simple glaucoma. It is the commonest form of glaucoma accounting for atleast half of all the glaucomas.

At least two of the three criteria in the presence of a normal open angle glaucoma confirmed by gonioscopy: [16]

1) An intraocular pressure above 21mm Hg on more than one occasion, asymmetry of IOP of more than 5 mm Hg between the two eyes and a circadian variation in IOP more than 8mm Hg

2) Optic nerve head changes suggestive of glaucomatous damage

3) Typical glaucomatous visual field

A. PATHOGENESIS

A sustained increase in IOP may be due to difficulty in its exit. Increased IOP is mainly due to increased resistance to the circulation of the aqueous at the pupil and /or to its drainage through the angle of the anterior chamber. Uveoscleral outflow accounts for 20% which is insufficient to maintain normal IOP.

1) CHANGES IN TRABECULAR MESHWORK[17]

I) Foreign materials such as glycosaminoglycans, amorphous material, extracellular lysosomes, plaque-like materials and proteins causing obstruction of the trabecular meshwork.

II) The functions of trabecular endothelial cells such as phagocytosis and synthesis and degradation of macromolecules is interfered.

III) Giant vacuoles present in the inner wall of endothelium of Schlemm's canal is lost. These vacuoles provide a pathway for drainage of fluid from meshwork into the Schlemm's canal.

IV) The endothelial cells are underactive or overwhelmed by foreign material, leading to cell death and loss of normal phagocytic activity, that is, the self-clearing filter property of the meshwork.

V) Decreased permeability of trabecular meshwork due to

a) Increased sensitivity to adrenergic agonists

b) Increased levels of gamma-globulin and plasma cells in trabecular meshwork and increased antinuclear antibodies

c) Altered corticosteroid metabolism

- Elevated plasma levels of cortisol
- Increased suppression of plasma cortisol with different doses of exogenous dexamethasone
- Disturbed pituitary adrenal function
- Increased inhibition of mitogen stimulated lymphocyte transformation by glucocorticoids.

Myocilin (TIGR-trabecular meshwork-inducible glucocorticoid response) gene governs the steroid responsiveness in POAG patients.[18]

2) CHANGES IN OPTIC NERVE HEAD

The local characteristics of the nerve head that play a role in resistance against increased IOP -

Diameter of scleral ring

Strength of lamina cribrosa

Integrity of vascular supply

Vasogenic theory of nerve damage [19]

This theory implies that structural and functional defects occurring in optic nerve head with glaucoma are due to ischemia.

Increased IOP leads to reduced capillary blood flow due to

a)Mechanical compression of vessels at lamina cribrosa

b)Reduced flow in annulus of Zinn which supplies nutrition to laminar and post laminar optic nerve head

Recently, Anderson put forth the hypothesis that inhibition of autoregulation of blood supply to optic nerve can cause increased susceptibility of disc to pressure induced ischemia.[20]

Mechanical theory of nerve damage

Lamina cribrosa cannot withstand high intraocular pressure. The nerve fibres are supported by glial tissue and have to bend over the edge of the disc.

Increased IOP leads to mechanical pressure on lamina cribrosa, altering capillary blood flow and reduced axoplasmic flow in the initial

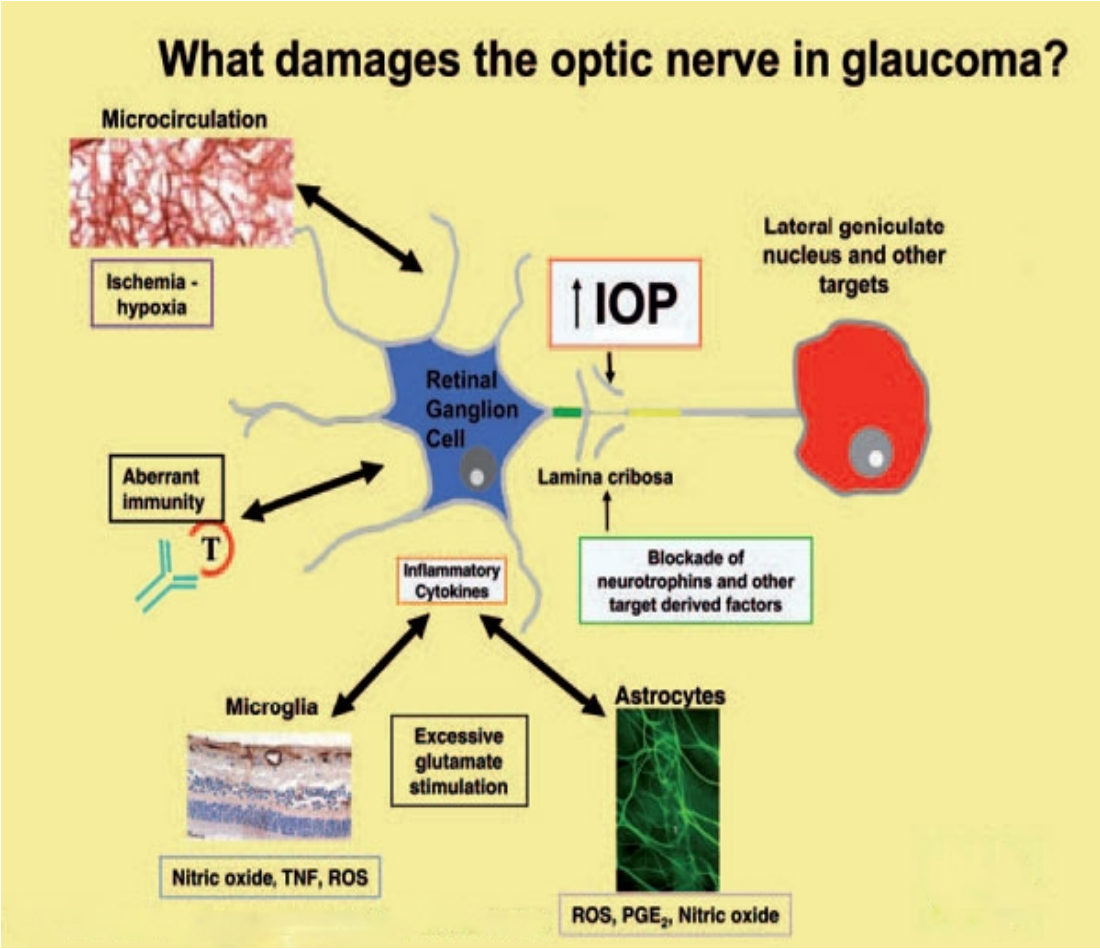
stages. Later, significant backward displacement and compaction of the laminar plates narrows the openings through which the axons pass, directly damaging the nerve fibre bundles, leading to atrophy.

Biochemical theory

Decrease in neurotropic factors / increased levels of neurotoxins.

Genetics

25 loci have been linked with POAG but only three genes have been identified –Myocilin, [21] Optineurin and WDR36.



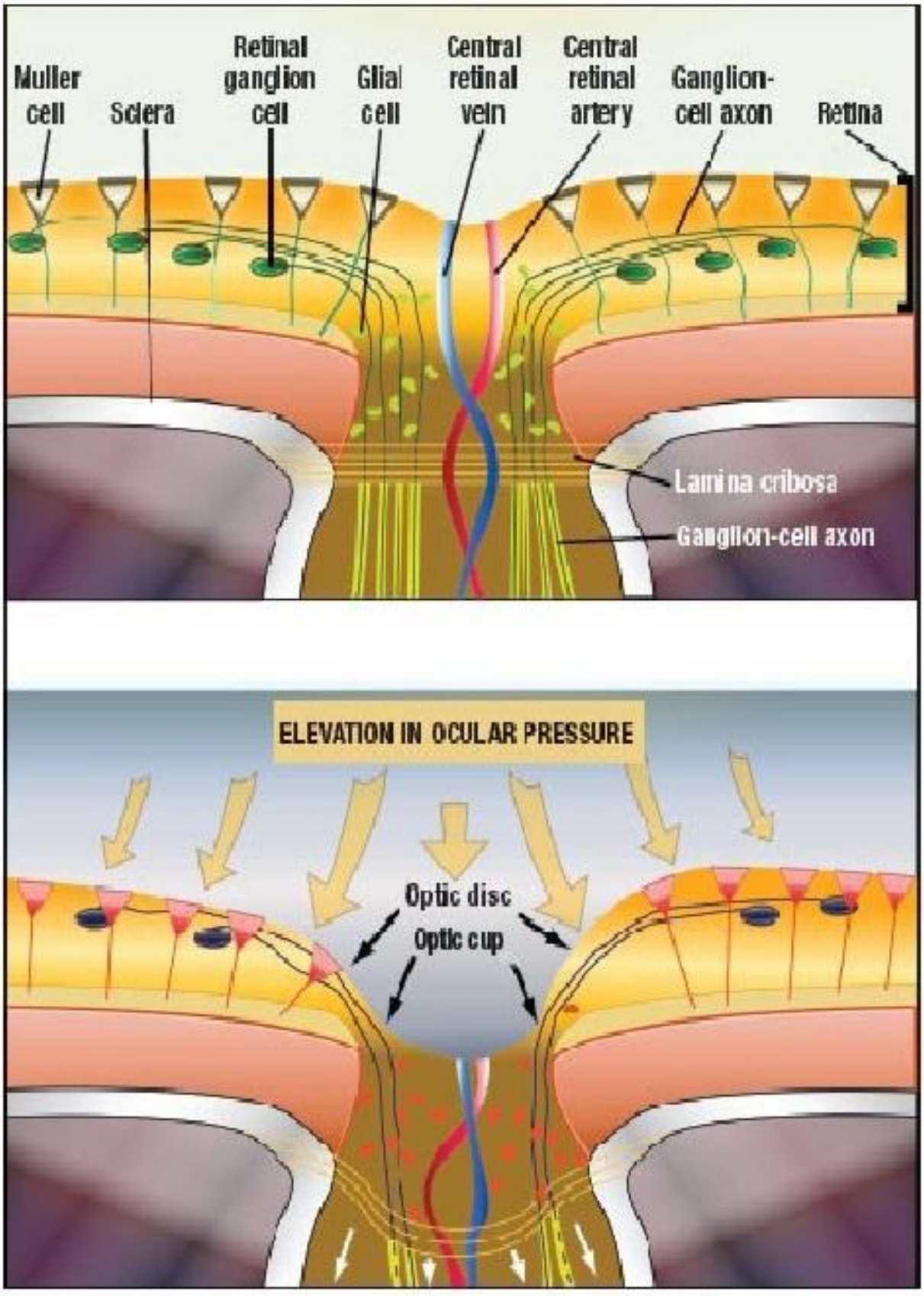


Fig.1. Pathogenesis of optic nerve head changes in glaucoma

B. CLINICAL FEATURES

Symptoms

POAG has an insidious onset, slowly progressive, bilateral condition usually asymptomatic until the advanced stages of the disease. Rarely the patient may notice a scotoma when performing a monocular visual task or may have frequent change of glasses. As glaucoma advances, they may become symptomatic from loss of fixation in one or both eyes or loss of peripheral vision to tubular vision, which interferes with activities such as driving.

Signs

1) Elevated IOP

Elevated IOP may range from 22 to 40 mmHg , occasionally may reach 60 or 80 mm Hg.

Normal diurnal fluctuation is less than 5 mm Hg while more than 8 mm Hg is abnormal. IOP is maximum in the early morning and minimum in the night.

Diurnal intraocular pressure measurements is useful in diagnosing POAG, explaining progressive damage inspite of apparent adequate IOP control. It helps in evaluating the efficacy of therapy and distinguishing normal tension glaucoma from POAG.[22]

Tonometer

Tonometer is the instrument used to measure the intraocular pressure by relating a deformation of the globe to the force responsible for the deformation. The two basic types of tonometers differ according to the shape of the deformation: indentation and applanation (flattening).

1.Applanation instruments

Variable force

The force required to flatten a standard area of the cornea is measured . The prototype in this group is the Goldmann applanation tonometer.

- Goldmann applanation tonometer (GAT), Perkins tonometer, Draeger tonometer, Mackay – Marg and Tono – pen tonometers, Pneumatic tonometer, Non- contact tonometer (NCT) / Ocular Response Analyzer and Ocuton tonometer

Variable area

The area of the cornea flattened by a known weight is measured.

- Maklakov tonometer is the prototype.

2.Indentation instruments

The shape of deformation is the truncated cone. Conversion tables must be used to measure the IOP.

Schiotz tonometer and Impact rebound tonometer.

The Schiotz tonometer is the prototype.

	APPLANATION	NON-CONTACT	SCHIOTZ
ADVANTAGES	1) not affected by ocular rigidity or stretchability of the globe	1) quick and may be delegated 2) no anaesthetic required 3) minimal risk of infection so safely used in post operative eyes[23]	1)Cheap and portable 2) Can be done on supine position 3) Screening purposes
DISADVANTAGES	1) need for anaesthetic 2) cannot be delegated 3) contact with cornea (slight chance of abrasion) 4) affected by central corneal thickness	1) uncomfortable to some patients 2) expensive 3) difficult to obtain reading on scarred corneas	1)Heavy(total weight 16.5g) 2) Corneal abrasions more likely 3) Risk of infection 4) Effect of scleral rigidity on reading 5)steep or thick cornea cause increased displacement fluid, hence it causes false high reading

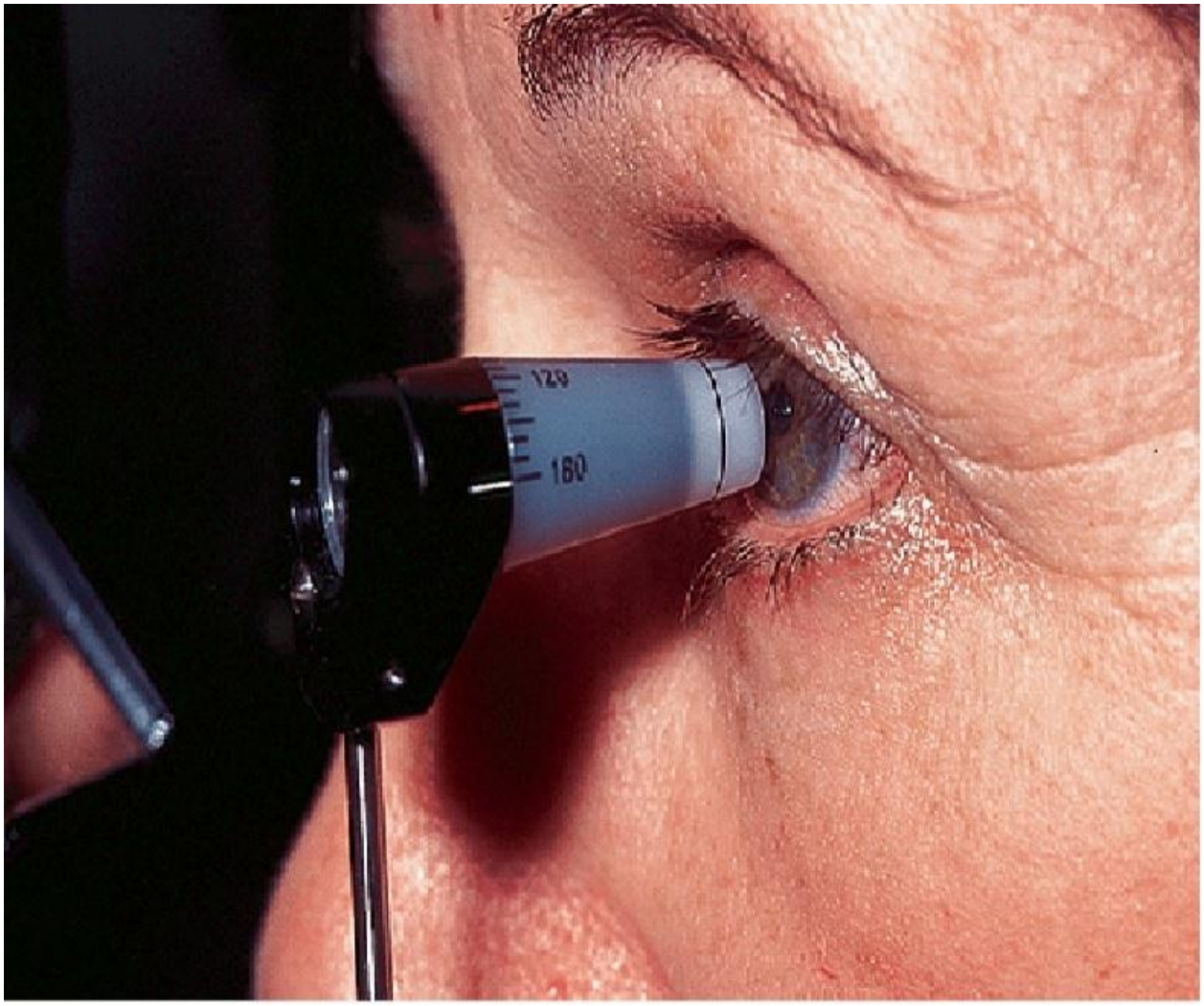


Fig.2. Goldmann applanation tonometry

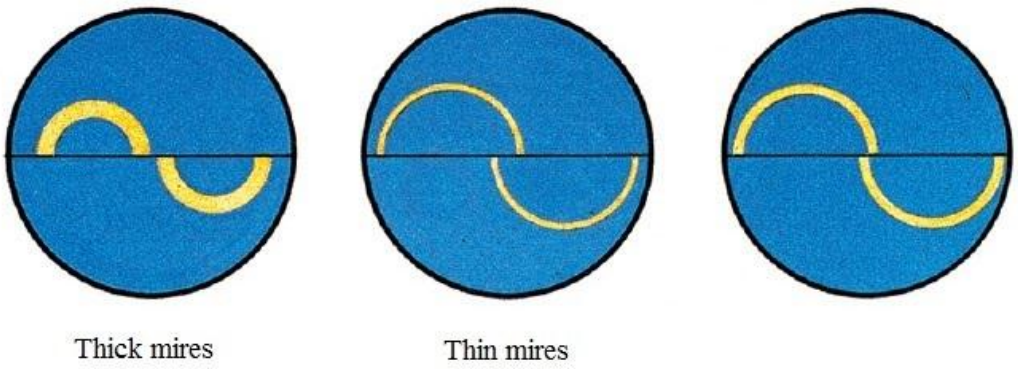


Fig.3. Fluorescein-stained mires during applanation tonometry

2) Optic disc changes

As bundles of axons are destroyed in an eye with glaucoma, the neural rim begins to thin in one of the several patterns.

a) Focal atrophy –The vertical cup-disc ratio becomes more than the horizontal cup disc ratio. The changes in chronological order are :

a. Polar notching (focal notching or pit like change (pseudopit) - usually in the inferior temporal quadrant, sharpened polar neural edge, sharpened rim and notching upto the disc margin.

b.Bayonetting sign - Sharp bend of the retinal vessels at the disc edge in the areas of sharpened rim.

b) Concentric atrophy

Enlargement of the cup in concentric circles, most often directed inferotemporally or superotemporally.

Temporal unfolding – the loss of neural rim tissue begins temporally and then progresses circumferentially toward the poles.[24]

The thinned out neural rim is seen as cresentic shadow adjacent to the disc margin .

c) Deepening of the cup

leads to overpass cupping and exposure of underlying lamina cribrosa. (Laminar dot sign)

d) Pallor/Cup discrepancy

Cupping greater than pallor indicates glaucomatous optic atrophy and pallor greater than cupping indicates non-glaucomatous optic atrophy.

e) Advanced glaucomatous cupping – loss of all neural tissue.

Bean-pot cupping – white disc with total loss of neural rim tissue and the vessels bend at the margin of the disc.[25]

Vascular signs

1) Optic disc haemorrhages[26]

- Splinter haemorrhages near the margin of the optic disc
- common location is in the inferior quadrant
- may be the first sign of glaucomatous damage preceding retinal nerve fibre layer defects, notches in the neural rim and field defects

2) Tortuosity of retinal vessels

is seen in advanced glaucomatous optic atrophy

3) Location of retinal vessels in relation to the cup

- Overpass cupping
- Baring of the circumlinear vessels

4) Nasal displacement of the retinal vessels does not provide a useful diagnostic parameter.

Peripapillary changes

Peripapillary atrophy consists of two zones – inner zone beta which is a depigmented chorio scleral crescent and outer zone alpha with increased

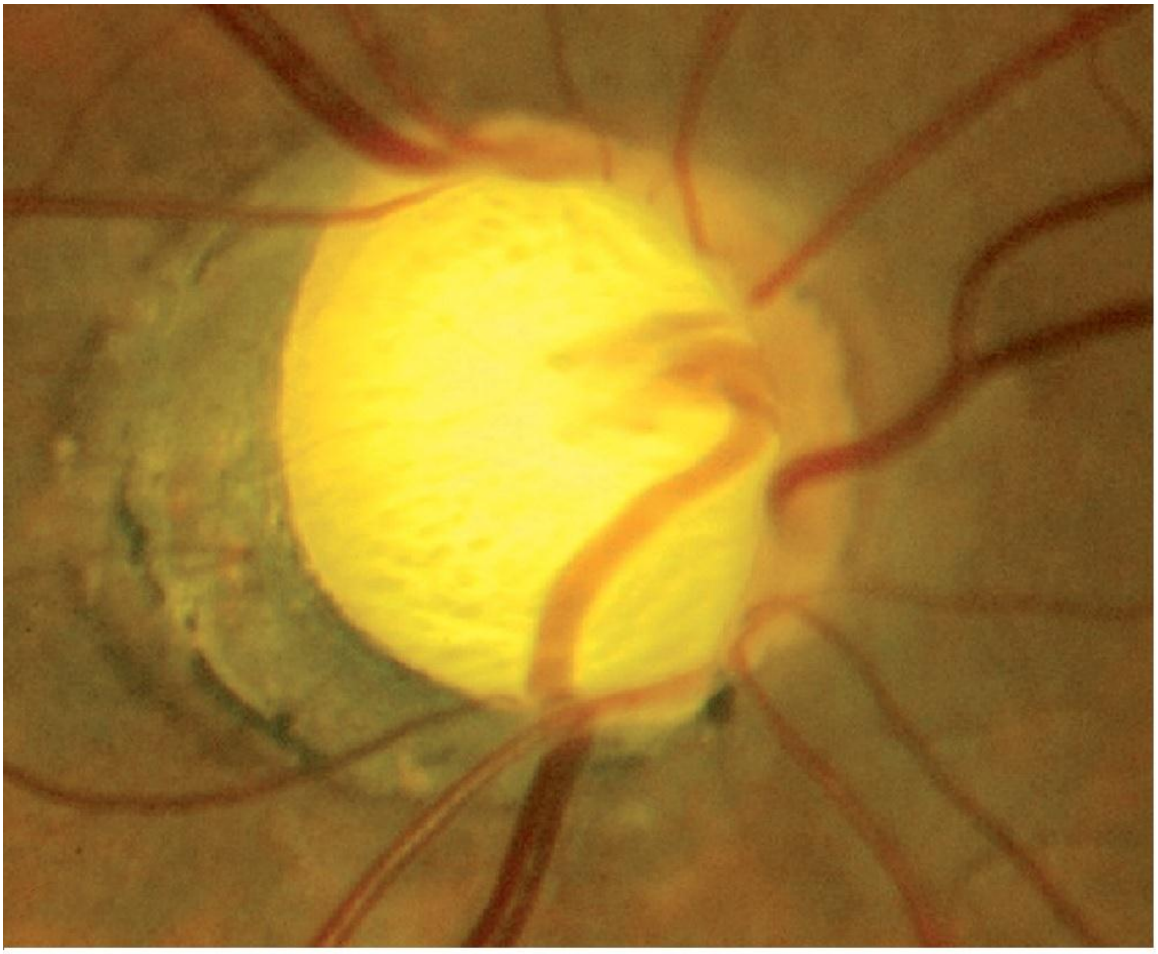


Fig.4. Glaucomatous optic disc

Fig.5. Retinal nerve fibre layer defect- red free filter

pigmentation. Zone beta is more associated with glaucoma and progressively increases in size with progression of glaucoma.[27]

Nerve fibre bundle defects

Appear as dark stripes or wedge shaped defects or diffuse loss of striations. The diffuse loss is more common in glaucoma patients than in ocular hypertensives. [28]

3) Gonioscopy

- This is performed using an indirect goniolens of either the Goldmann or Zeiss 4 mirror type.

- In POAG, anterior chamber angle is open.

- Have more iris processes, higher insertion of the iris root, more trabecular meshwork pigmentation [29] and a greater than normal degree of segmentation in the pigmentation of the meshwork.

4) Visual field abnormalities

It is initially observed in Bjerrum area, $10\text{-}25^0$ from fixation. Later, it ranges from paracentral scotomas, nasal step, Seidel scotoma, arcuate or Bjerrum scotoma, ring scotoma or double arucate scotoma, tubular vision to end-stage or near total defect, with only a residual temporal island of vision.

The nonspecific changes are generalised depression of visual field, concentric contraction of the visual field which is more marked in the nasal

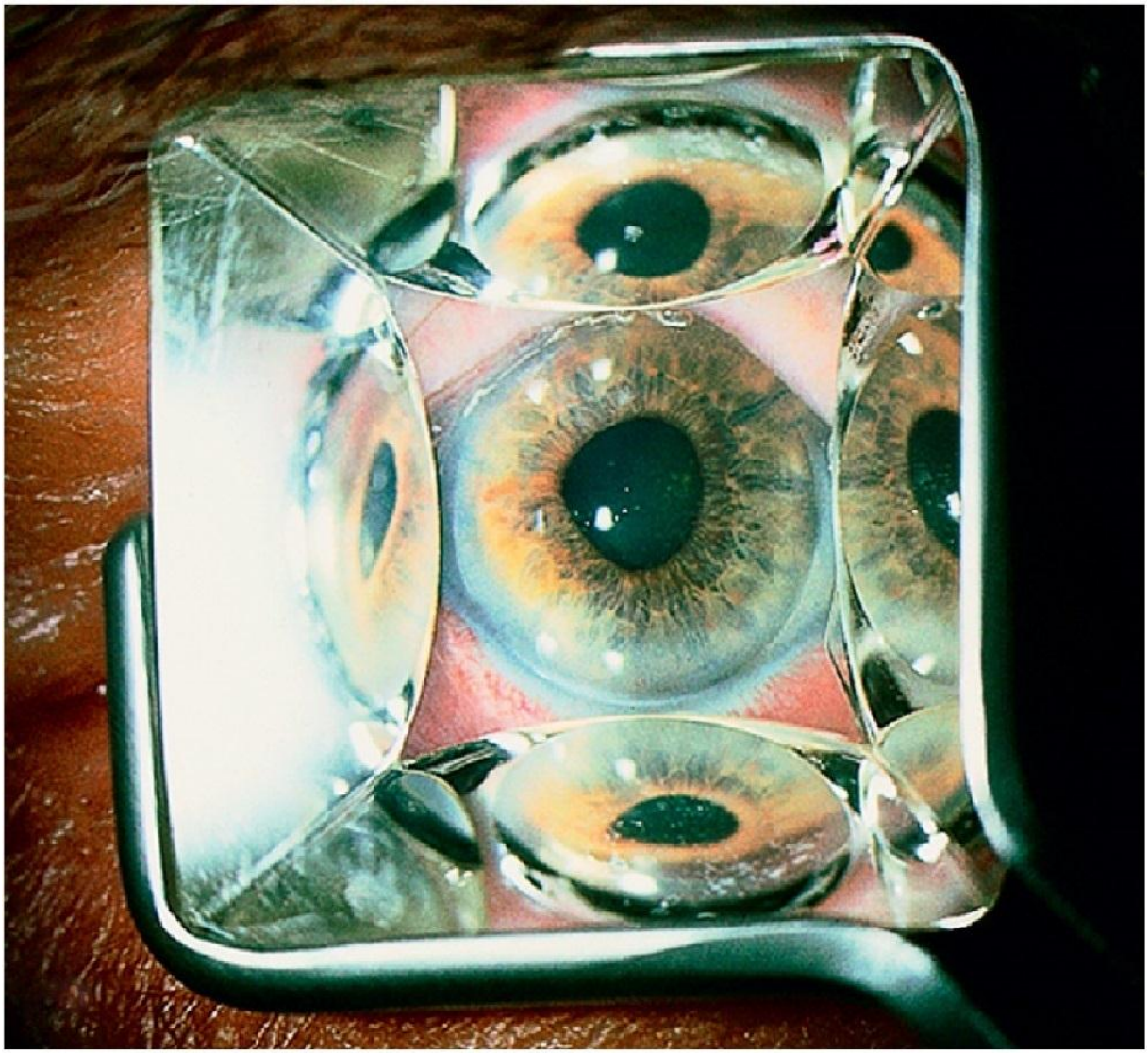


Fig.6. Zeiss four mirror gonioscopy

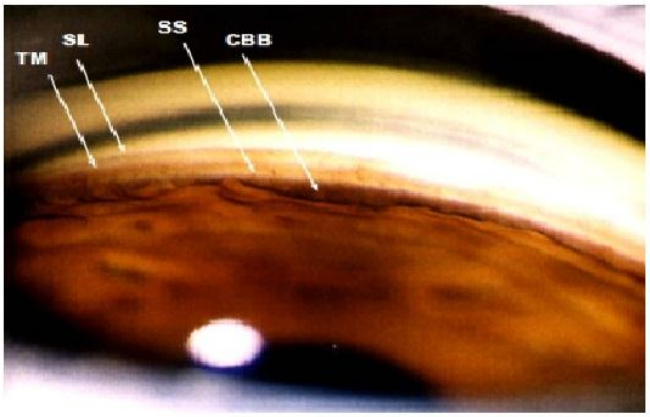


Fig.7. Gonioscopy showing open angles

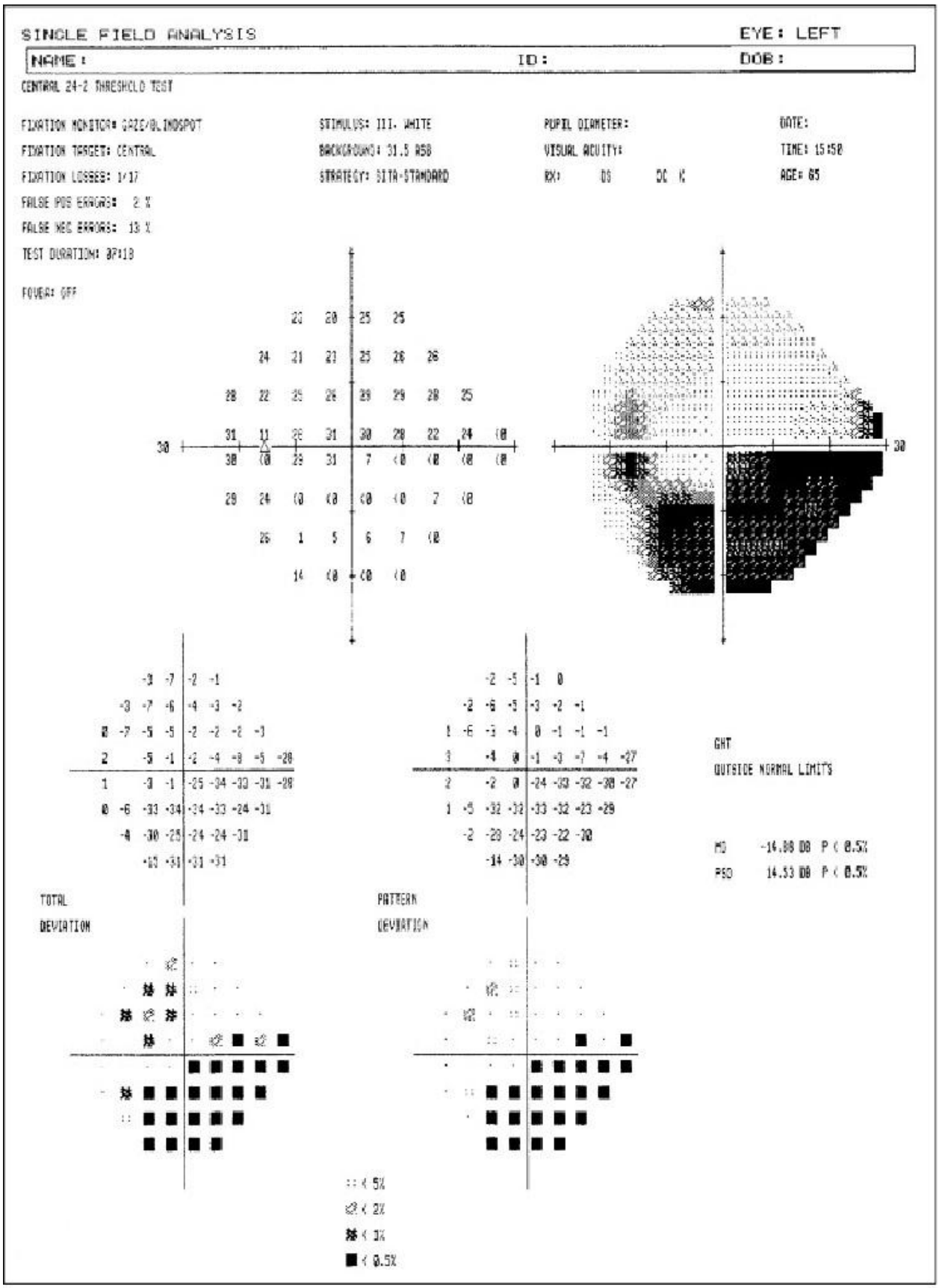


Fig.8. Humphrey perimetry showing inferior arcuate scotoma

field called “crowding of the peripheral nasal isoptres”, enlargement of the blind spot and angioscotoma.

Progressive visual field loss is the most useful guide for diagnosis, treatment and follow up in POAG. [30]

C.DIAGNOSIS

A diagnosis of POAG can be made after performing the following tests:

1. Intraocular pressure recording
2. Optic nerve head / retinal nerve fibre layer (RNFL) assessment
3. Gonioscopy
4. Visual field analysis

Optic Nerve Head Assessment is done using

- Slitlamp and an auxiliary fundus lens (Goldmann 3 mirror contact lens, the handheld 78 D or 90D lens, Hruby lens slitlamp attachment)

- A diagrammatic representation of the disc, neuroretinal rim, vascular alterations and nerve layer defects at every follow-up

- Stereo photography of the optic nerve head (ONH) -to ascertain small changes sequentially

Analysis of Optic nerve head and Retinal nerve fibre layer is done using

- Direct ophthalmoscope with a red-free filter (ophthalmoscopy)

- Slitlamp and an auxiliary fundus lens with a red-free filter

- Glaucoma diagnosis (GDx) RNFL analyzer uses the principle of Confocal Scanning Laser Polarimetry. It is used to measure the peripapillary RNFL thickness.

- Optical Coherence Tomography provides high resolution cross-sectional imaging of the ONH , RNFL and macula. It gives the best axial resolution. The macular imaging programme detects early glaucomatous changes.

- Heidelberg Retina Tomography uses the principle of confocal scanning laser ophthalmoscopy. It is used to obtain three-dimensional images of optic disc to detect glaucomatous damage and to assess progression in glaucoma.

Perimetry

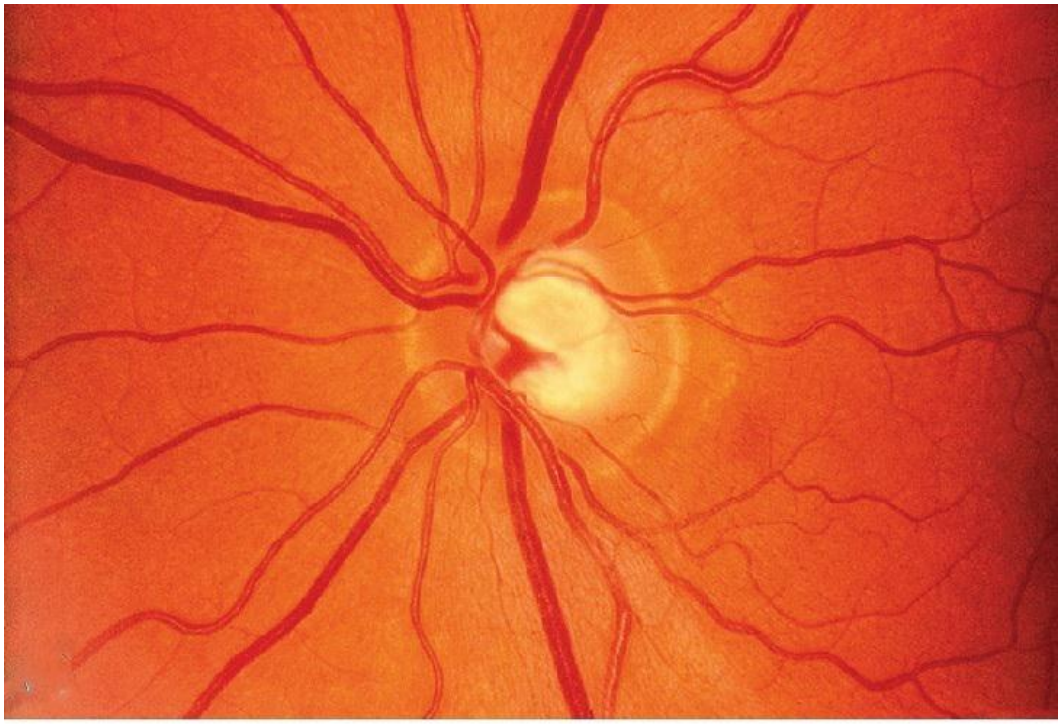
Perimetry is the technique employed to examine and quantify the visual field using targets of various sizes and colours.

It is of two types

1) Kinetic
2) Static

Static techniques

Automated (Humphrey Field Analyzers HFA , Octopus) and manual (Goldmann perimetry) are examples of static techniques. It is the preferred method of testing field which uses various testing strategies.[31]



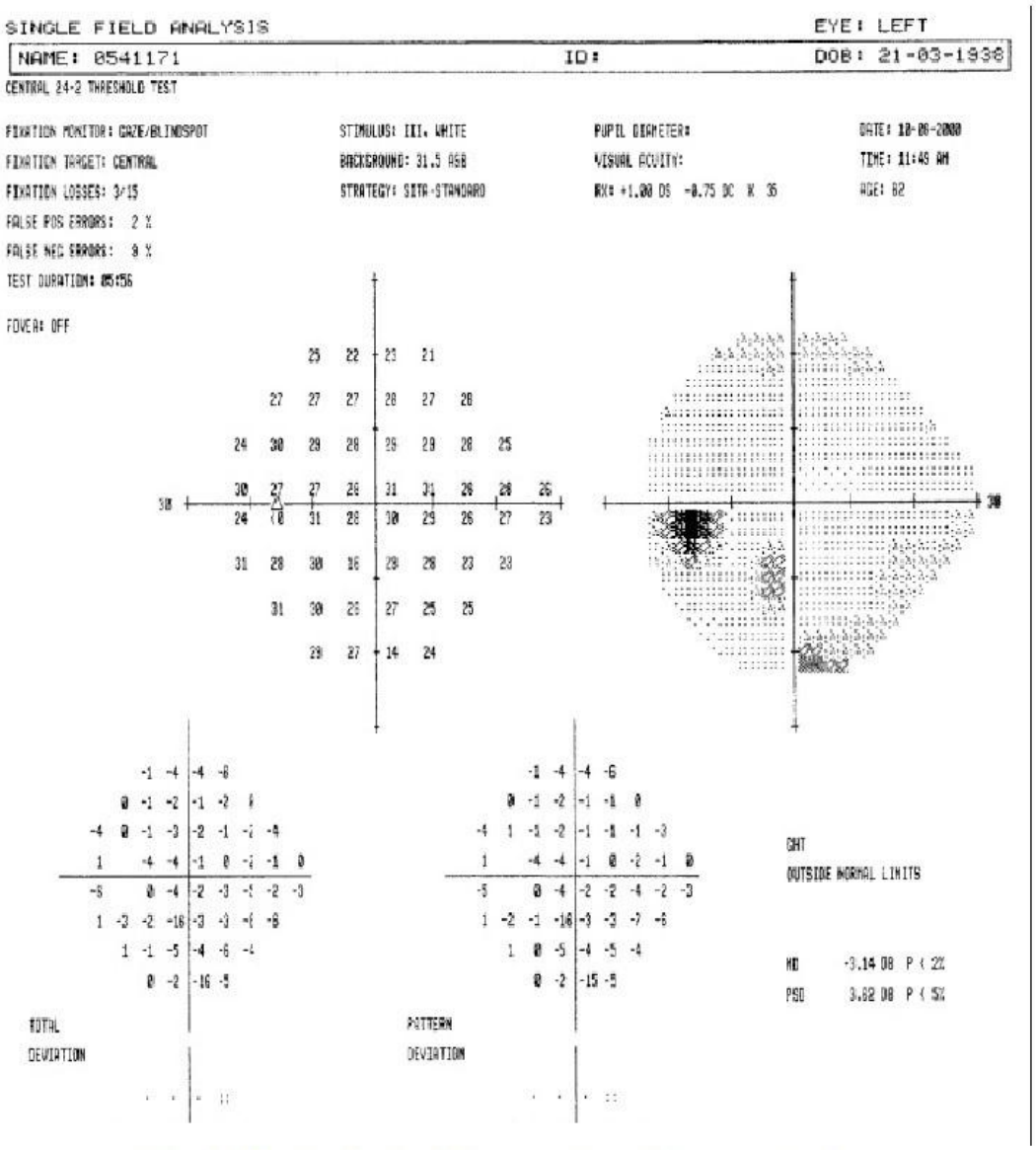
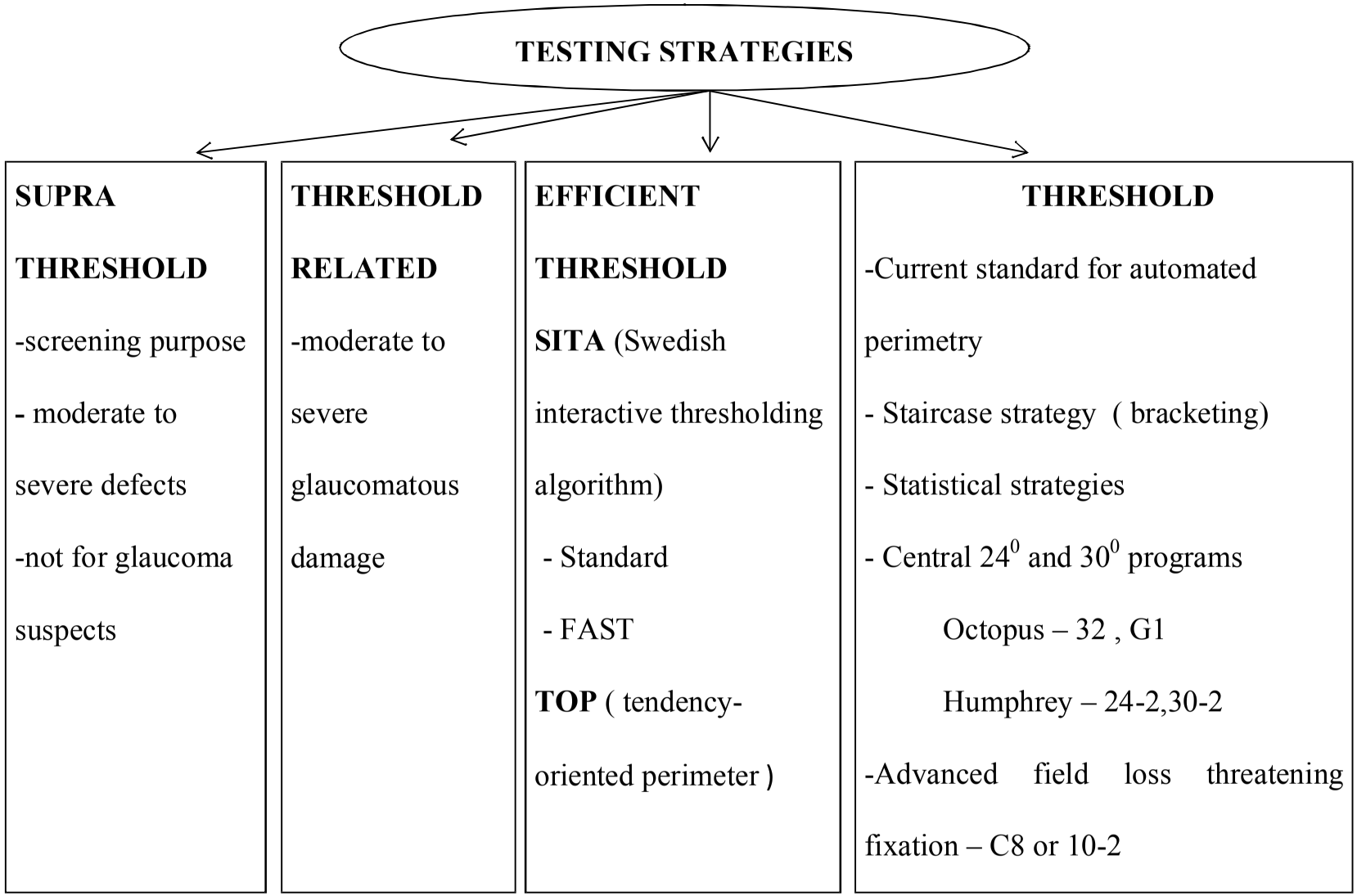


Fig.9. Minimal optic disc cupping with paracentral scotoma


TESTING STRATEGIES
SUPRA THRESHOLD
-screening purpose
- moderate to severe defects
-not for glaucoma suspects
THRESHOLD RELATED
-moderate to severe glaucomatous damage
EFFICIENT THRESHOLD
SITA (Swedish interactive thresholding algorithm)
- Standard
- FAST
TOP (tendency-oriented perimeter)
THRESHOLD
-Current standard for automated perimetry
- Staircase strategy (bracketing)
- Statistical strategies
- Central 24^0 and 30^0 programs
Octopus – 32 , G1
Humphrey – 24-2,30-2
-Advanced field loss threatening fixation – C8 or 10-2

Kinetic techniques

Confrontation, Tangent screen , Lister perimeter and Goldmann perimeter are the examples of kinetic perimetry in which the intensity and size of the stimulus is kept constant but the stimulus location is moved (non-seeing to a seeing area).

Newer perimetric techniques

1) Short-wavelength automated perimetry (SWAP)

-blue on yellow perimetry

-helps in early identification of glaucomatous damage by testing small ganglion cells, called bistratified blue-yellow ganglion cells - available on HFA II (700 series) and Octopus 1-2-3[32]

2) Frequency – doubling technology (FDT) perimetry

- Low spatial frequency sinusoidal grating undergoing rapid phase - reversal flicker

- Preferentially activate M cells early identification of glaucomatous damage[33]

- older instrument using 16 to 18 large test fields.

- screening programmes

- new instrument Matrix with 54 smaller test fields

3) HRP – High Pass Resolution Perimetry, also known as ring perimetry

4) Flicker perimetry in the Octopus perimeter

For assessing possible progression

Delta program with the Octopus perimeter

Humphrey Field Analyzer

- STATPAC 2 (includes linear regression analysis and glaucoma change probability)
- Progressor Program for analysis of serial fields
- Glaucoma Change Probability (GCP)
- Glaucoma Probability Analysis (GPA)

D.MANAGEMENT

IOP is the only modifiable risk factor in POAG. Hence all treatment modalities target the IOP. Lowering IOP is associated with significant lowering of glaucoma progression[34].

Steps towards efficient treatment of POAG includes –

1.Assessment of glaucomatous damage [35]

	Disc	Visual field
Mild	0.0 – 0.5 with uniform pink rim	None, mild depression, or slight defect
Moderate	0.6-0.7 with some local narrowing of rim	General depression, arcuate defect,or paracentral scotoma
Advanced	0.8- 0.9 with rim narrowing or notching	Large arcuate, double arcuate, hemifield loss, or fixation threatened

2.Fix target IOP

Target IOP

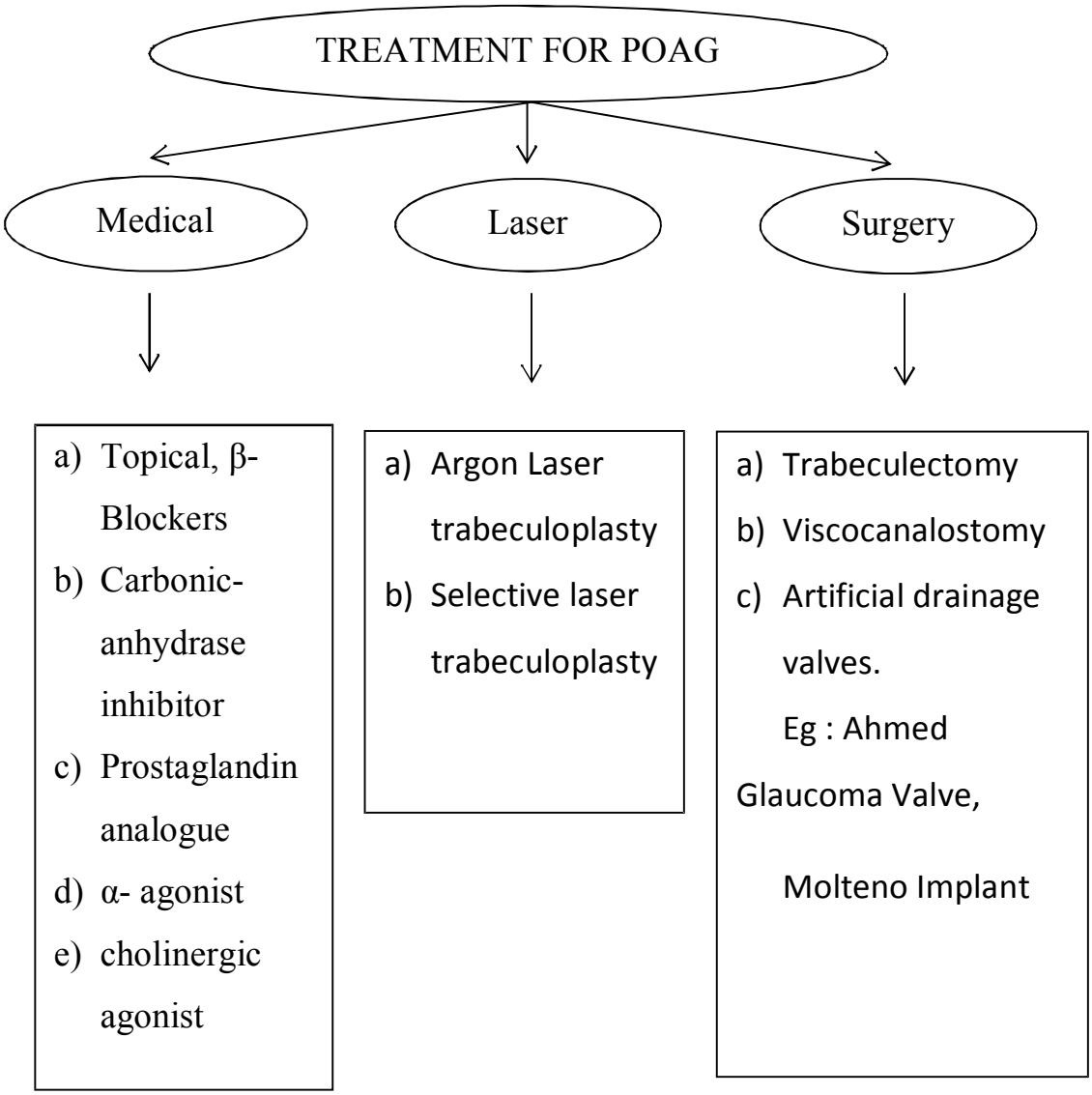
It is defined as " A range of acceptable IOP levels within which the progression of glaucomatous neuropathy will be halted / retarded ."[36]

Lower target IOP set if lower the initial IOP, older age, more advanced glaucomatous damage presence of cardiovascular disease or diabetes, the risk of central retinal vein occlusion, as well as individuals who are one- eyed or in whom visual fields or disc evaluation is not possible.

	TARGET IOP	
	% IOP reduction	IOP set below (mm Hg)
Mild	20	18
Moderate	30	15
Advanced	50	13

3.Medical / laser / surgery to achieve target IOP

4.Follow up to look for progression of glaucomatous damage



1) Medical therapy

The basic principle of medical therapy is

- to use the least amount of medicine that will control the glaucoma with the fewest side effects.

- to treat only one eye initially in symmetric cases, so the fellow eye can be used as a control in determining the efficacy of therapy.

Drug Classification	Mechanism of Action	IOP Reduction	Side Effects	Contraindications
Prostaglandin analogues	Increase uveoscleral and/or trabecular outflow	25%–33%	conjunctival hyperaemia, hypertrichosis, iris and periocular hyperpigmentation ,exacerbation of herpetic keratits, uveitis, cystoid macular edema,	Macular edema History of herpetic keratitis
Beta-adrenergic antagonists (beta-blockers)	Decrease aqueous production	20%–25%	Corneal toxicity bronchospasm (nonselective), bradycardia, depression, Impotence	Chronic obstructive pulmonary disease (nonselective) bradycardia, hypotension
Para sympathomimetic agents	Increase trabecular outflow	20%–25%	Myopic shift,brow ache, cataract,retinal detachment, epiphora,paradoxical angle closure	Neovascular, uveitic, or malignant glaucoma

Drug Classification	**Mechanism of Action**	**IOP Reduction***	**Side Effects**	**Contraindications**
Alpha-adrenergic agonists	Nonselective: improve aqueous outflow Selective: decrease aqueous production; increase uveoscleral outflow	20%–25%	Conjunctival injection, allergic reactions Fatigue ,Headache	Monoamine oxidase inhibitor therapy Infants and children younger than 2 years
Carbonic anhydrase inhibitors	Decrease aqueous production	15%–20%	Metallic taste, allergic conjunctivitis corneal edema, Stevens-Johnson syndrome, malaise, anorexia, electrolyte imbalance, renal calculi, blood dyscrasias	Sulfonamide allergy Kidney stones Aplastic anaemia Thrombocytopenia Sickle cell disease

Neuroprotective agents

These include anecortave, cannabinoids, cellular cytoskeletal modulators (ethacrynic acid latrunculins), olmesartan, lomerizine, neurotropins, memantine, nitric oxide, prostanoid agents and rho kinase inhibitors.

2) Laser

The Glaucoma Laser Trial[37] provided some support for laser trabeculoplasty (argon laser) as initial therapy. But, mostly it is used as an adjunct to medical therapy. Argon, diode, or selective laser energy (selective laser trabeculoplasty) is applied to the surface of the trabecular meshwork to increase the aqueous outflow.[38]

3) Surgical intervention

Indications

a. Patients who are poor candidates for conventional medical treatment

b. Patients in whom the target IOP is unlikely to be achieved with topical medications alone

c. Further progression of visual field loss likely to affect the patient's quality of life

d. When rapid IOP lowering to the desired target level is required in patients with rapidly progressive glaucomatous optic neuropathy where quality of life would otherwise suffer

e. whenever there is progressive glaucomatous damage despite "maximum tolerable medical therapy"

f. Patients with poor drug compliance or drug tolerance

g. Patients not accessible to an ophthalmologist

h. Good IOP control with surgery in the fellow eye

FILTERING SURGERY

Opening or fistula at the limbus → direct communication between the anterior chamber and the subconjunctival space → aqueous absorbed by surrounding tissues or crosses conjunctival epithelium and drained through tears.

In patients with glaucoma that is refractory to standard filtering surgery, aqueous drainage devices can be considered. It is indicated in patients with extensive conjunctival scarring, chronic ocular inflammation and ocular trauma. Glaucoma drainage devices are not as effective as filtering surgery in controlling IOP.

Cyclophotocoagulation can be considered as the last resort for the patients with refractory glaucomas, those with multiple failed filtering procedures and with visual potential is poor.[39]

<table>
<tr><th>FISTULIZING
TECHNIQUES</th><th>NONPENETRATING
PROCEDURES</th></tr>
<tr><td rowspan="3">1.Partial thickness
Trabeculectomy
2.Full thickness
Sclerectomy
Trephination
Thermal sclerostomy
Iridencleisis</td><td>1.Deep sclerectomy
2.Viscocanalostomy</td></tr>
<tr><td>GLAUCOMA DRAINAGE
IMPLANTS</td></tr>
<tr><td>Open-tube drainage devices
-Baerveldt , Molteno,
Schocket
Flow-Resticted drainage devices
-Ahmed, Krupin</td></tr>
</table>

GLAUCOMA SUSPECT

Open angle by gonioscopy and one of the following in at least one eye[40] :

1) IOP consistently >21 mm Hg by applanation tonometry

2) Appearance of the optic disc or retinal nerve fibre layer suggestive of glaucomatous damage

3) Diffuse or focal narrowing or sloping of the disc rim

4) Diffuse or localized abnormalities of the RNFL , especially at superior and inferior poles

5) Disc haemorrhage

6) Asymmetric appearance of the disc or rim between fellow eyes, suggesting loss of neural tissue

7) Visual fields suspicious of early glaucomatous damage

DECISION TO TREAT IN GLAUCOMA SUSPECTS WITH ELEVATED IOP

The patients are classified into low, moderate, or high risk for progression based on the available evidence and clinical judgement

High risk – Treatment must be initiated

Moderate risk – Treatment given if required, or monitor closely

Low risk – Monitor IOP , optic nerve structure and function, and treat if progression occurs

NORMAL TENSION GLAUCOMA

Normal tension glaucoma (NTG) is a progressive disease

- IOP consistently equal or less than 21mm on diurnal testing, with no single measurement greater than 24mm Hg and off treatment

- Open drainage angles on gonioscopy

- Absence of any secondary cause for a glaucomatous optic neuropathy

- Typical optic disc damage with glaucomatous cupping and loss of neuroretinal rim

- Visual field defect compatible with the glaucomatous cupping and loss of neuroretinal rim

- It is a disease of elderly and is more prevalent in females. OPA 1 gene is the major genetic marker of NTG[41]

Etiology

The factors involved in the causation of NTG can be divided into :

a. Pressure independent factors

Abnormal blood flow – Vasospasm as in migraine and Raynaud's phenomenon

Nocturnal hypotension due to night dose antihypertensives

Abnormal blood coagulability and increased blood viscosity

Systemic diseases like diabetes mellitus, ischemic heart disease, carotid artery atheroma, cerebrovascular accidents

b. Pressure dependent factors

IOP is still a risk factor in the development and progression of the disease.

Characteristic features

There is increased incidence of

- optic disc haemorrhages
- peripapillary atrophy
- thin neuroretinal rim especially inferiorly and inferotemporally

- more localised RNFL defects, closer to the macula

- field defects tend to be localised, deeper and closer to fixation

Management

1. Detect and confirm damage

Serial perimetry confirms the existing field defects and detects progression.

2. Rule out high-tension glaucoma

This is done by repeated IOP measurements.

3. Detect / rule out etiological factors and risk factors

Look for evidence of

a) Vasospastic disorders such as migraine or Raynaud's phenomenon

b) Nocturnal dip in BP in elderly patients, wherever possible by 24 hours continuous BP monitoring

c) Rule out systemic haemodynamic abnormalities e.g. Myocardial dysfunction, hyperlipidaemia, hypertension and diabetes mellitus

4. Rule out neurological causes of disc pallor

CT scan or MRI of brain mandatory in the following situations:

- In patients who do not show disc/field correlation (pallor more than cupping)
- Visual field defects respecting the vertical midline

• In patients who have neurological signs and symptoms other than visual loss

5. Monitoring for deterioration progression

6. Treatment options

The treatment of NTG is directed at preventing further optic disc damage by modulating the pressure dependent and pressure independent factors.

• **IOP lowering treatments**

Reduction of IOP by 30% to halt or slow down progression[42]

This can be achieved by topical medications , argon laser trabeculoplasty (ALT) or by surgery.

- Prostaglandin and prostamide derivatives like latanoprost and bimatoprost.

- Patients who show progression and in whom the medical treatment does not achieve 30% reduction IOP need filtration surgery preferably with the use of anti-fibroblastic agents such as mitomycin-C and 5- flurouracil.

• **Non IOP lowering treatments**

oral calcium channel – increases ONH capillary perfusion

Topical betaxolol, brimonidine -neuroprotective agents if progression continues despite adequate lowering of IOP.

GLAUCOMA AWARENESS

1) Public

2) Health care personnel and eye care

3) Human resource development

1) PUBLIC

Talk to your family and friends about glaucoma

Can visit websites that are exclusive for glaucoma

Free educational booklets

National Eye Health Education Program (NEHEP) is a program to raise awareness about glaucoma among people at higher risk and their friends. Various public service announcements through radio, television and print are made in this.

2.HEALTH CARE PERSONNEL AND EYE CARE [43]

To increase eye care personnels, ophthalmologists, optometrist, equipment technician. National and local training centers must be increased.

1.**Primary**

-Comprises promotive and preventive actions carried out by the ophthalmic assistants. Referral of the cases done at this level.

- Social and community developments which promote health through changes in behaviour and the environment. This is the hardest to be achieved but has the greatest impact.

- Strengthening family and community cooperation for recognition and appropriate care of the glaucoma patients

- Delivery of eye care

2.Secondary

Carried out at district level which should provide definitive management . The main challenge at this level is case detection. Patient with possible disease is referred to the tertiary level. If filtering surgery is been done the patient must have periodic evaluation with the secondary health care worker and should be re-referred when IOP raises.

3.Tertiary

Variety of diagnostic and therapeutic measures

Training- Trained to perform iridectomies , gonioscopy to examine optic disc and visual fields.

Screening- Since entire population cannot be screened the high risk population can be focussed on.

Availability of drugs- Anti glaucoma medications must be available at all levels of health care.

Mobile eye services

These fulfil the functioning of delivering primary and secondary eye care. These services should be temporary and replaced by permanent infrastructure for eye health care.

3.HUMAN RESOURCE DEVELOPMENT

- To increase eye care personnel, ophthalmologists, optometrists, equipment technician.

- Local and national training centres must be increased.

CAMPAIGNS FOR GLAUCOMA AWARENESS

INTERNATIONAL

WHO Programme for the Prevention of Blindness and Deafness is to provide essential eye care to all populations and to eliminate avoidable blindness.

World Glaucoma Association (WGA) works to optimize the awareness of glaucoma through cooperation among regional and national Glaucoma Societies.

The World Glaucoma Patient Association (WGPA)works through national Glaucoma Patient Associations.

"BIG – Beat Invisible Glaucoma" campaign – the 6th World Glaucoma Awareness Week 2014 is to be held on March 9-15, 2014 to raise the awareness of glaucoma and the importance of regular eye exams for early detection of glaucoma. During this week, patient and eye care professionals around the world participate in the activities to support the cause.

January is glaucoma awareness month.

NATIONAL

Glaucoma society of India works to -

- Glaucoma Society of India

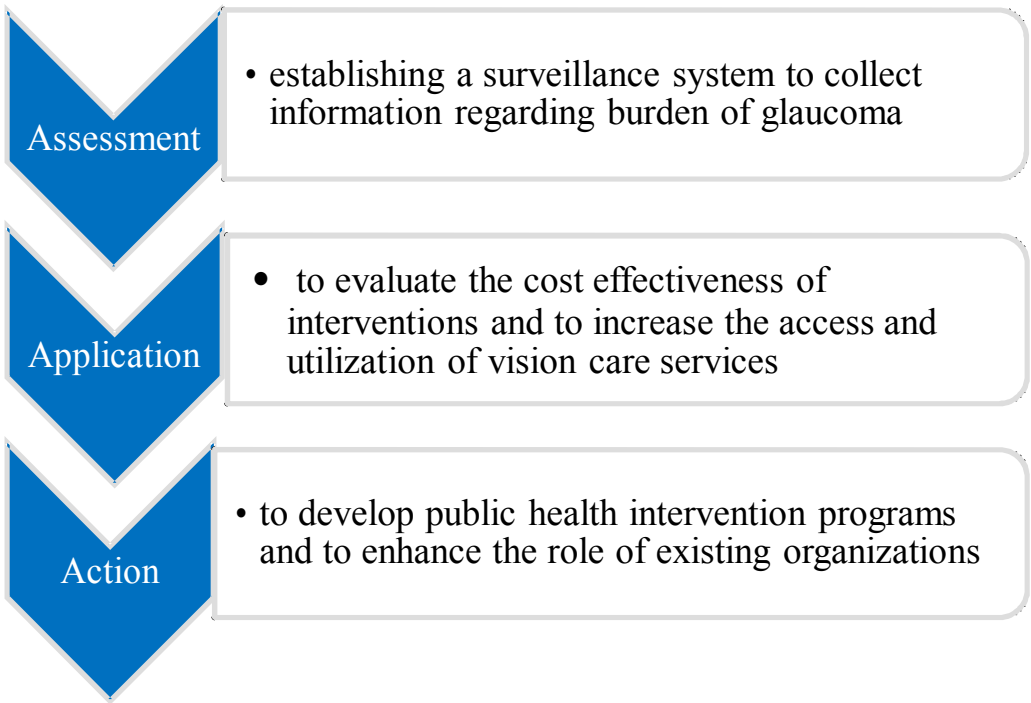
to create awareness among the public

to guide glaucoma patients for the correct treatment.

- Glaucoma India Education Program to enlighten the advances in the diagnosis and treatment of glaucoma to as many as Ophthalmologists as possible.

- Vision 2020

Strategies to improve vision health



VISION 2020

What Vision 2020 has done regarding glaucoma

- Research into various aspects of glaucoma is being conducted internationally.

- New treatment options are evaluated and new anti-glaucoma drugs have been on trial.

- Screening tests for glaucoma have been developed.

What Vision 2020 is doing regarding glaucoma

-Training of ophthalmologists, optometrist and mid-level personnel, including refractionist.

-The secondary and tertiary eye care centres are equipped to provide essential eye care services for the glaucoma patients.

- Availability of basic equipment and low cost drugs.

- Glaucoma screening made a part of regular eye checks especially for patients at high risk.

- Increase public awareness about glaucoma and about the consequences of the disease been left undiagnosed.

- Patient education and counselling for increasing patient drug compliance and regular eye checks.

REVIEW OF LITERATURE

The prevalence of glaucoma suspects on the basis of elevated IOP in persons older than 40 years was 4% to 10% in the studies conducted by Bankes JL, et al.[44]Rates of ocular hypertension reported in the Andhra Pradesh Eye Disease Study (APEDS)[45] was 0.42% , 1.1% in the Aravind Comprehensive Eye Survey (ACES)[46], 3.08% in Vellore Eye study(VES).[47]

Primary open angle glaucoma

The prevalence of POAG is 0.5 - 1% in persons aged over 40 in the studies performed in U.S and Western Europe.[48] In the Tajimi study[49], 3.9% of those over 40 years had POAG with majority of people having IOP less than 21mmHg. In pooled analysis of population based studies, prevalence was seen to increase from 0.6% (40-49 years) to 1.5% (50 – 59 years), 2.7% (60-69 years), 5.1% (70 – 79 years) and 7.33% in those above 80 years. The Los Angeles Latino Eye Study[50] found that Latinos in the United States have a prevalence of 4.7 % . A hospital based study by Smita et al [51] in Northern India showed a high prevalence of POAG (33%).

The VES reported the lowest rates of 0.41%, mainly because the study was limited to the age group of 30-60years and low rate of visual field performance. 2.56% in APEDS, 1.7% in ACES, 1.62% in Chennai Glaucoma Study (CGS) rural, 3.51% in CGS[52] urban and 2.99% in West Bengal Glaucoma Study (WBGS).[53]

Increasing age was a risk factor in all studies. Males were at greater risk in the ACES. No such difference was noted by any of the other studies. Myopia was a risk factor only in ACES.

A positive correlation between glaucoma and diabetes was shown by Rotterdam study, Netherlands[54] and the Blue Mountains eye study, Australia. The Baltimore eye survey found little evidence of an association between glaucoma and either insulin dependent or non-insulin dependent diabetes.

There was no significant association between hypertension and POAG in the study by Tielsch et al[55] and Uhm and Shin[56]. Population based data from Framingham study and Baltimore eye survey also failed to find any association between BP and POAG. The Rotterdam study reported an association of systemic hypertension with high-tension glaucoma. A hospital based study by Mohammed et al [57] showed positive correlation between POAG and systemic hypertension.

65% of those with POAG in APEDS, 45% in ACES, 67% in CGS (rural) and 82% in CGS (urban) had normal presenting IOP. This means that single normal IOP does not rule out the disease. Optic disc evaluation is necessary to identify those with glaucoma.

IOP measured with GAT and NCT was compared in a study by Shalini Mohan et al. IOP was comparable at lower range but was unreliable in

patients with higher IOP range.[58] In a study by Muller et al, there was no significant difference between GAT and NCT while indentation tonometer showed differences.[59]

The mean vertical cup disc ratio (CDR) was 0.56 in VES done in an unselected population[60] and 0.39 in CGS. In a study by Krishna et al, the CDR and the rim disc ratio was considered to be clinically significant in determining abnormal glaucomatous optic discs.[61]

According to Kasner et al, the absence of peripapillary atrophy(PPA) is associated with decreased risk of glaucomatous damage in ocular hypertensives.

The proportion of persons bilaterally blind from POAG was 11% (APEDS), 1.6% (ACES), 5.2% in WBGS, 3.2% (CGS rural) and 1.5% (CGS urban).

The rate of undiagnosed patients was 92.6% in APEDS, 93% in ACES and 98.5% in CGS . 50% of the patients diagnosed to have POAG in ACES had an previous eye examination by the ophthalmologist but <20% of them were detected to have glaucoma before the study evaluation.

Awareness about glaucoma ranged from 0.27% in the rural population Andhra Pradesh (Krishnaiah et al)[62] to 13.3% (Sathyamangalam et al).[63]According to Dandona et al,[64] awareness rate was 2.3%. This is very much lower than the rates reported from United States of America (72-81%)

and Australia (70-92%). In a hospital based study by Prabhu et al, 4.8% were aware of glaucoma.

The knowledge about glaucoma was 8.7% in CGS, 3.1% in the study by Prabhu et al[65] and 5.6% in the study by Krishnaiah et al.

The awareness with respect to age, gender, religion was not significant in the study by Prabhu et al and Tenkir et al.[66]

The literacy status and glaucoma awareness were significantly associated in CGS and studies by Krishnaiah et al, Prabhu et al, Tenkir et al and Gasch et al.[67]

AIMS AND OBJECTIVES

To study the prevalence of primary open angle glaucoma in patients aged 40 and above attending Ophthalmology outpatient department in Tirunelveli Medical College Hospital to enable early detection of this silent vision killer.

To evaluate their awareness and knowledge about glaucoma and educate them regarding the disease which will infuse confidence in glaucoma patients to face life with full knowledge of the disease and follow up with involvement.

MATERIALS AND METHODS

One hundred patients aged 40 years and above attending Ophthalmology outpatient department in Tirunelveli Medical College Hospital from January 2012 to September 2013 were screened for primary open angle glaucoma.

Institutional Ethical Committee approval was obtained before starting first patient enrolment. A convenient sample size of hundred was considered.

The study design was prospective cross-sectional study using random sampling. The study was randomized by choosing 1 among 10 patients using block randomization technique to prevent selection bias. By this method of using randomised table 100 patients were chosen from 1000 patients. A written informed consent was obtained in the patients 40 years and above and they had their each eye tested for the following with the available facilities in our hospital :

1. Distant visual acuity using Snellen's chart
2. Near vision using Times Roman near vision chart
3. Refraction by autorefractometer and subjective correction
4. Intraocular measurement using
    - Schiotz indentation tonometer
    -Goldmann applanation tonometer
    - Non contact tonometer

5. Optic disc evaluation using 90 D lens in slit lamp

6. Visual field analysis - central 30^0 using Octopus 300 automated perimeter – TOP programme[68]

7. Gonioscopy using Zeiss 4 mirror goniolens

A questionnaire was given to these patients to collect information regarding patient's awareness and knowledge about glaucoma. The questionnaire was translated in Tamil and back- translated to English. Literacy level of all subjects were obtained. The patients who were able to read **and** write any language were considered as literates.[69] The questionnaire was administered prior to the history and examination procedures for glaucoma. Details about previous eye check and attending eye camps were also obtained.Patients having heard of glaucoma even before the study were defined as aware and who had some understanding about the disease were defined as knowledgeable.

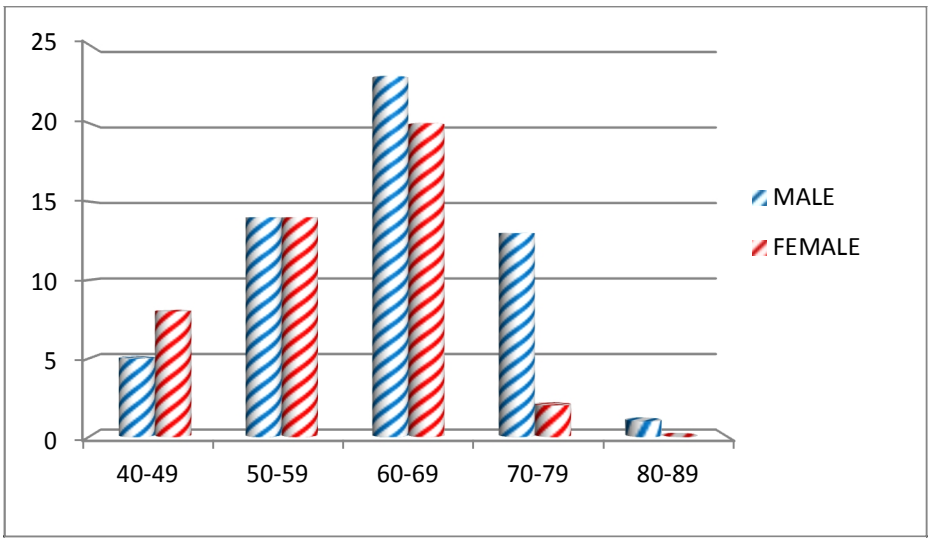
INCLUSION CRITERIA	Patients aged 40 and above
	Both males and females
EXCLUSION CRITERIA	Patients not willing for examination
	Known PLHA patients
	Patients with angle closure glaucoma

RESULTS

Table 1: Demographics of the study population

	MALE	FEMALE
40-49	5	8
50-59	14	14
60-69	23	20
70-79	13	2
80-89	1	0
TOTAL	56	44

Chart 1 : Demographics of the study population



The above table1 and chart 1 shows the percentage distribution in the study subjects according to their age and sex.There were 56 males and 44 females.

Table 2 : Details of age, sex, vision and intraocular pressure of the primary open angle glaucoma patients

S.NO	AGE	SEX	VISION		RE(mm Hg)			LE(mm Hg)		
			RE	LE	S	NCT	AT	S	NCT	AT
1	60	F	6/60	6/24	22.4	23	22	12.2	13	12
2	48	M	6/18	6/24	22.4	25	22	24.4	22	22
3	65	M	5/60	6/12	12.2	12	10	12.2	12	11
4	73	M	4/60	4/60	24.4	23	22	26.6	33	26
5	46	M	6/12	6/9	19.6	13	14	14.6	14	14

The above table 2 shows the age, sex, vision and intraocular pressure values of the 5 primary open angle glaucoma patients.

Table 3 : Details of CDR, PPA and visual field of the primary open angle glaucoma patients

PATIENT	CDR		PPA	FIELD	
	RE	LE		RE	LE
1	0.6	0.9	+	Superior arcuate scotoma	Tubular field
2	0.4	0.4	-	Paracentral scotoma	Normal
3	0.9	0.5	+	Temporal island of vision	Superior arcuate Scotoma
4	0.6	0.9	+	Inferior arcuate scotoma	Tubular field
5	0.7	0.4	+	Double arcuate scotoma	Normal

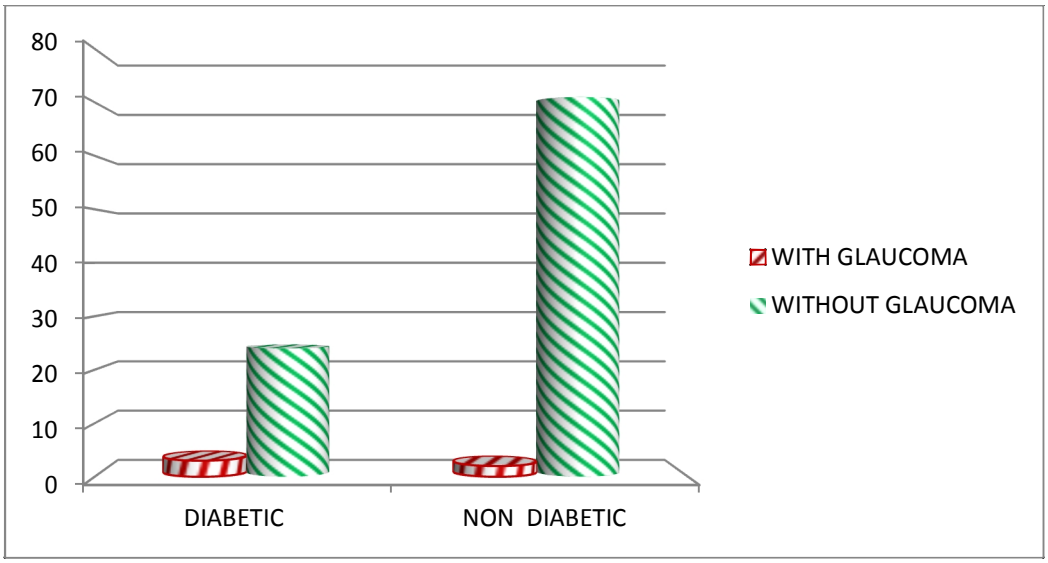
Chart 2 : Correlation between cup disc ratio and visual field defect

The above chart 2 shows a linear relationship between CDR and visual field defects.

Table 4: Diabetes and primary open angle glaucoma patients

	GLAUCOMA	
	+	-
DIABETICS	3	24
NON DIABETICS	2	71

Chart 3: Diabetes and primary open angle glaucoma patients

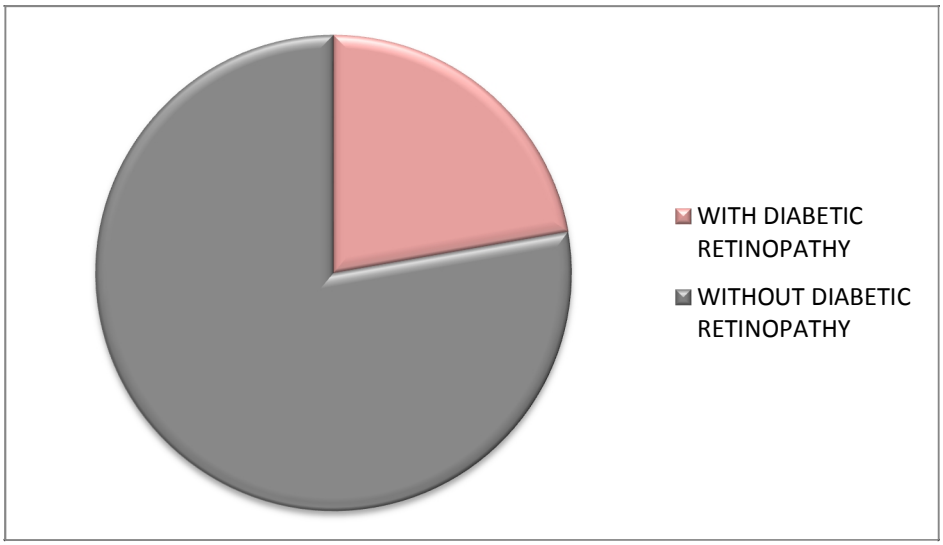


The above table 4 and chart 3 shows the association between diabetes and primary open angle glaucoma. By Fisher's Exact Probability test, no significant association (p =0.120) was found between the diabetic status and the occurrence of POAG. 3 out of 27 diabetics were diagnosed to have POAG.

Table 5: Diabetic retinopathy among diabetics

DIABETIC RETINOPATHY	
+	-
6	21

Chart 4: Diabetic retinopathy among diabetics

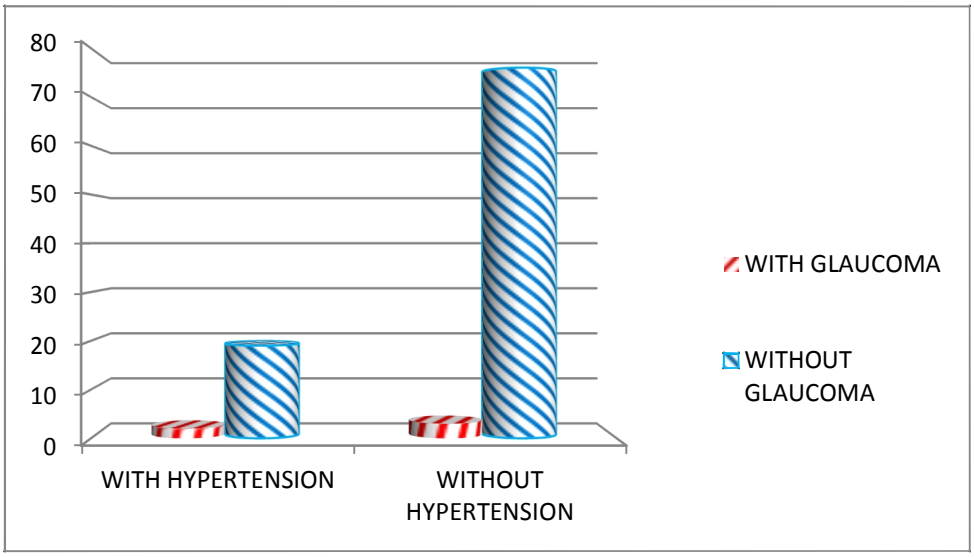


The above table 5 and chart 4 shows the percentage of diabetic retinopathy among the diabetics. Among the diabetics, 6 out of 27 diabetics had diabetic retinopathy .

Table 6: Primary open angle glaucoma among the hypertensives

		GLAUCOMA	
		+	-
HYPERTENSION	+	2	19
	-	3	76

Chart 5: Primary open angle glaucoma among the hypertensives



The above table 6 and chart 5 shows the association between hypertension and POAG. By Fisher's Exact Probability test, no significant association (p =0.282) was found between the hypertensive status and the occurrence of POAG. There were 21 hypertensive patients. 2 of the 21 hypertensive patients had POAG.

Table 7: Comparison of intraocular pressure measured with applanation and non contact tonometer in the right eye:

	MEAN (mm Hg)	SD	P
GAT	14.22	3.37	0.133
NCT	14.50	3.38	

The above table 7 shows that the mean IOP in right eye was 14.22 ± 3.37 mm Hg with applanation tonometer and 14.50 ±3.38 mm Hg with non contact tonometer. Using paired sample student's T test, it was found that there was no significant difference (p =0.133) between GAT and NCT in the right eye.

Table 8: Comparison of intraocular pressure measured with applanation and Schiotz tonometer in the right eye:

	MEAN (mm Hg)	SD	P
GAT	14.22	3.37	0.000
SCHIOTZ	15.41	3.19	

The above table 8 shows that the mean IOP in right eye was 14.22 ± 3.37 mm Hg with applanation tonometer and 15.41± 3.19 mm Hg with Schiotz tonometer. Using paired sample student's T test, it was found that there was a significant difference (p<0.001) between GAT and Schiotz in the right eye.

Table 9: Comparison of intraocular pressure measured with applanation and non contact tonometer in the left eye:

	MEAN(mm Hg)	SD	P
GAT	13.87	3.35	0.001
NCT	14.45	3.56	

The above table 9 shows that the mean IOP in the left eye was 13.87 ±3.35 mm Hg with GAT and 14.45 ±3.56 mm Hg with non contact tonometer. Using paired sample student's T test, it was found that there was a significant difference (p 0.001) between GAT and NCT in the left eye.

Table10:Comparison of intraocular pressure measured with applanation and Schiotz tonometer in the left eye:

	MEAN(mm Hg)	SD	P
GAT	13.87	3.35	0.000
SCHIOTZ	15.22	3.18	

The above table 10 shows that the mean IOP in the left eye was 13.87 ±3.35mm Hg with GAT and 15.22 ± 3.18mmHg with Schiotz. Using paired

sample student's T test, it was found that there was a significant difference between GAT and Schiotz tonometer ($p < 0.001$).

Table 11: Mean cup disc ratio in the right eye of normal and primary open angle glaucoma patients

	MEAN	SD	P
NORMAL	0.31	0.05	0.000
POAG	0.64	0.18	

The above table 11 shows the mean CDR in the right eye of normal and primary open angle glaucoma patients. The mean CDR in the right eye was 0.31 in the normal patients and 0.64 in the POAG patients. Using unpaired t test, it was found that there was a significant difference ($p < 0.001$) between the CDR of glaucomatous and non glaucomatous patients in the right eye.

Table 12: Mean cup disc ratio in the left eye of normal and primary open angle glaucoma patients

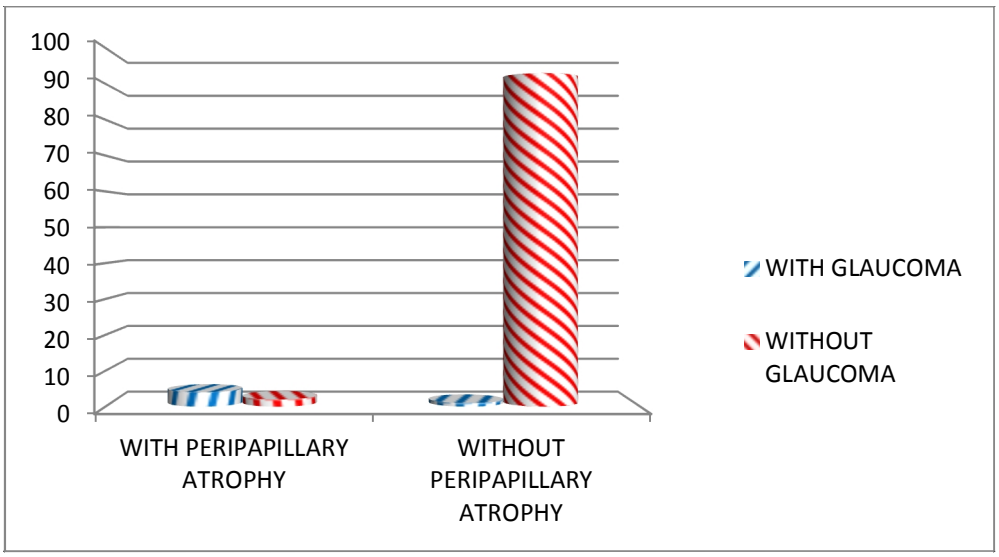
	MEAN	SD	P
NORMAL	0.32	0.05	0.000
POAG	0.62	0.25	

The above table 12 shows the mean CDR in the left eye of normal and primary open angle glaucoma patients. The mean CDR in the left eye was 0.32 in the normal patients and 0.62 in the POAG patients. Using unpaired t test, it was found that there was a significant difference ($p < 0.001$) between the CDR of glaucomatous and non glaucomatous patients in the left eye.

Table 13: Peripapillary atrophy and primary open angle glaucoma

		GLAUCOMA	
		+	-
PERIPAPILLARY ATROPHY	+	4	2
	-	1	93

Chart 6: Peripapillary atrophy and primary open angle glaucoma

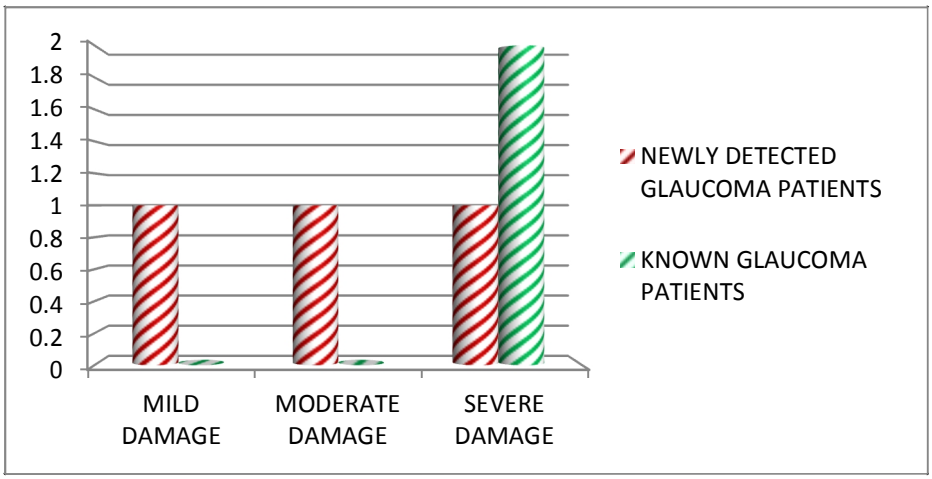


The above table 13 and chart 6 shows the association between peripapillary atrophy and POAG. With Fisher's Exact Probability test with Yates continuity correction for small expected numbers, p value was found to be <0.001. This means that there is significant association between peripapillary atrophy and POAG. Out of the 6 patients who had peripapillary atrophy, 4 had POAG.

Table 14: Severity of glaucomatous damage and glaucoma patients

SEVERITY OF DAMAGE	GLAUCOMA PATIENTS	
	NEWLY DETECTED	KNOWN
MILD	1	0
MODERATE	1	0
SEVERE	1	2

Chart 7: Severity of glaucomatous damage and glaucoma patients

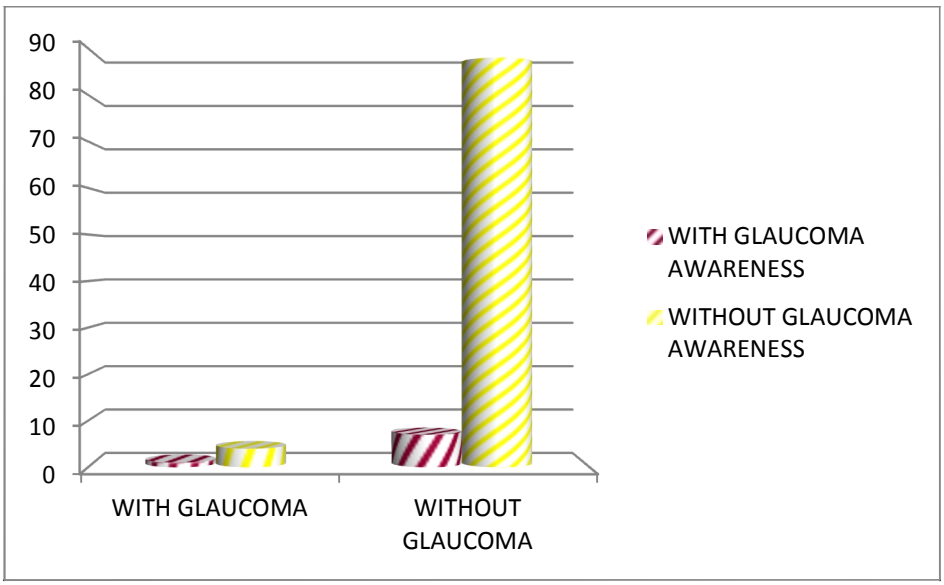


The severity of glaucomatous damage is shown in the table 14 and chart 7. Among the newly diagnosed POAG patients, 1 had mild,1 had moderate and the other had severe glaucomatous damage. Both the known POAG patients had severe glaucomatous damage.

Table 15: Glaucoma awareness among the patients

		GLAUCOMA AWARENESS	
		+	-
GLAUCOMA	+	1	4
	-	7	88

Chart 8: Glaucoma awareness among the patients



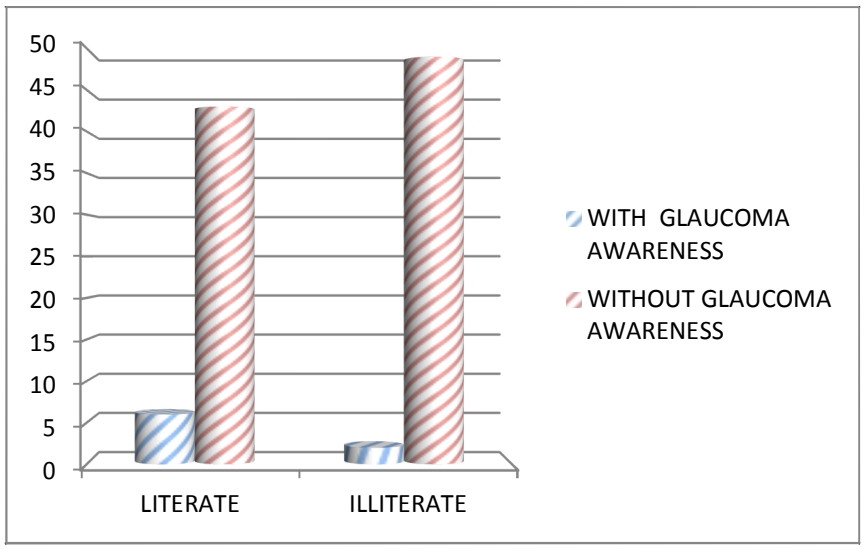
The above table 15 and chart 8 shows the glaucoma awareness among the patients. With Fisher's Exact Probability test with Yates continuity correction for small expected numbers, p value was found

to be 0.866.This means that there is no significant association between the presence of glaucoma and glaucoma awareness,only 8 were aware of glaucoma of which 1 had POAG.

Table 16: Glaucoma awareness and literacy

		GLAUCOMA AWARENESS	
		+	-
LITERACY	+	6	43
	-	2	49

Chart9 : Glaucoma awareness and literacy



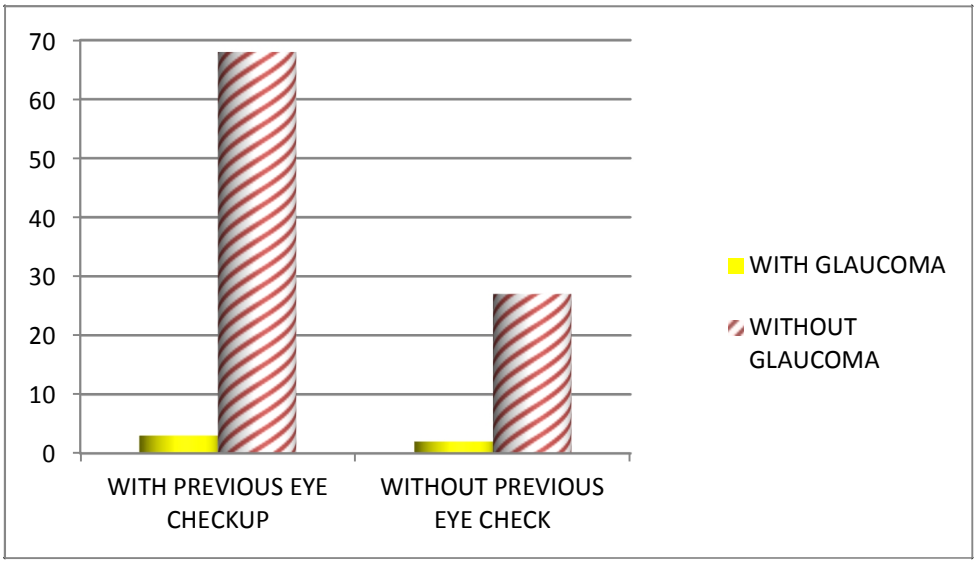
The above table 16 and chart 9 shows the association between glaucoma awareness and literacy. With Fisher's Exact Probability test, p value was found to be 0.122.This means that there is no significant

association between literacy and glaucoma awareness.6 out of 49 literates and 2 out of 51 illiterates were aware of glaucoma.

Table 17: Previous eye check up and primary open angle glaucoma

		GLAUCOMA	
		+	-
PREVIOUS EYECHECK	+	3	68
	-	2	27

Chart 10: Previous eye check up and primary open angle glaucoma



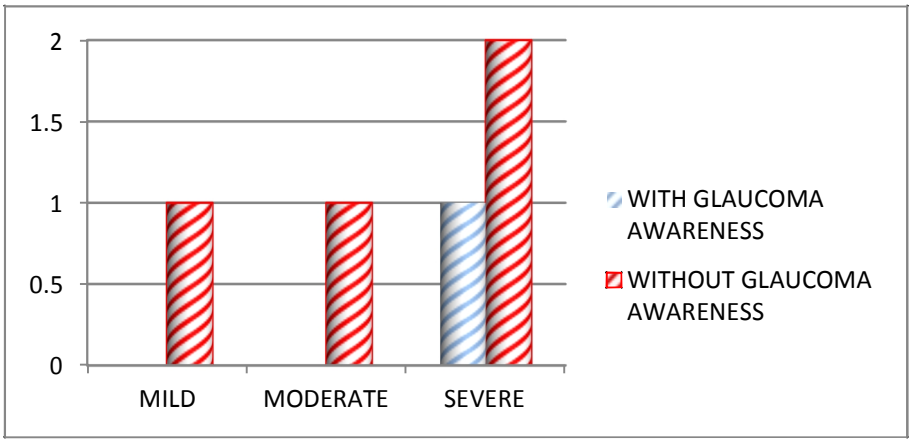
The above table 17 and chart 10 shows the association between previous eye check up and glaucoma. With Fisher's Exact Probability test, p value was found to be 0.453. It means that there is no significant association between POAG and previous eye check . 71 patients had previous ocular

examination, of which 3 had POAG. 2 of the primary open angle glaucoma patients diagnosed in our study did not have their eyes checked before.

Table 18: Glaucomatous damage and glaucoma awareness

	GLAUCOMA AWARENESS	
	+	-
MILD	0	1
MODERATE	0	1
SEVERE	1	2

Chart 11: Glaucomatous damage and glaucoma awareness



The above table 18 and chart 11 shows the association between glaucomatous damage and awareness. With Fisher's Exact Probabilitytest,value was found to be 0.659. This means that there is no significant association between the severity of glaucoma and glaucoma awareness.

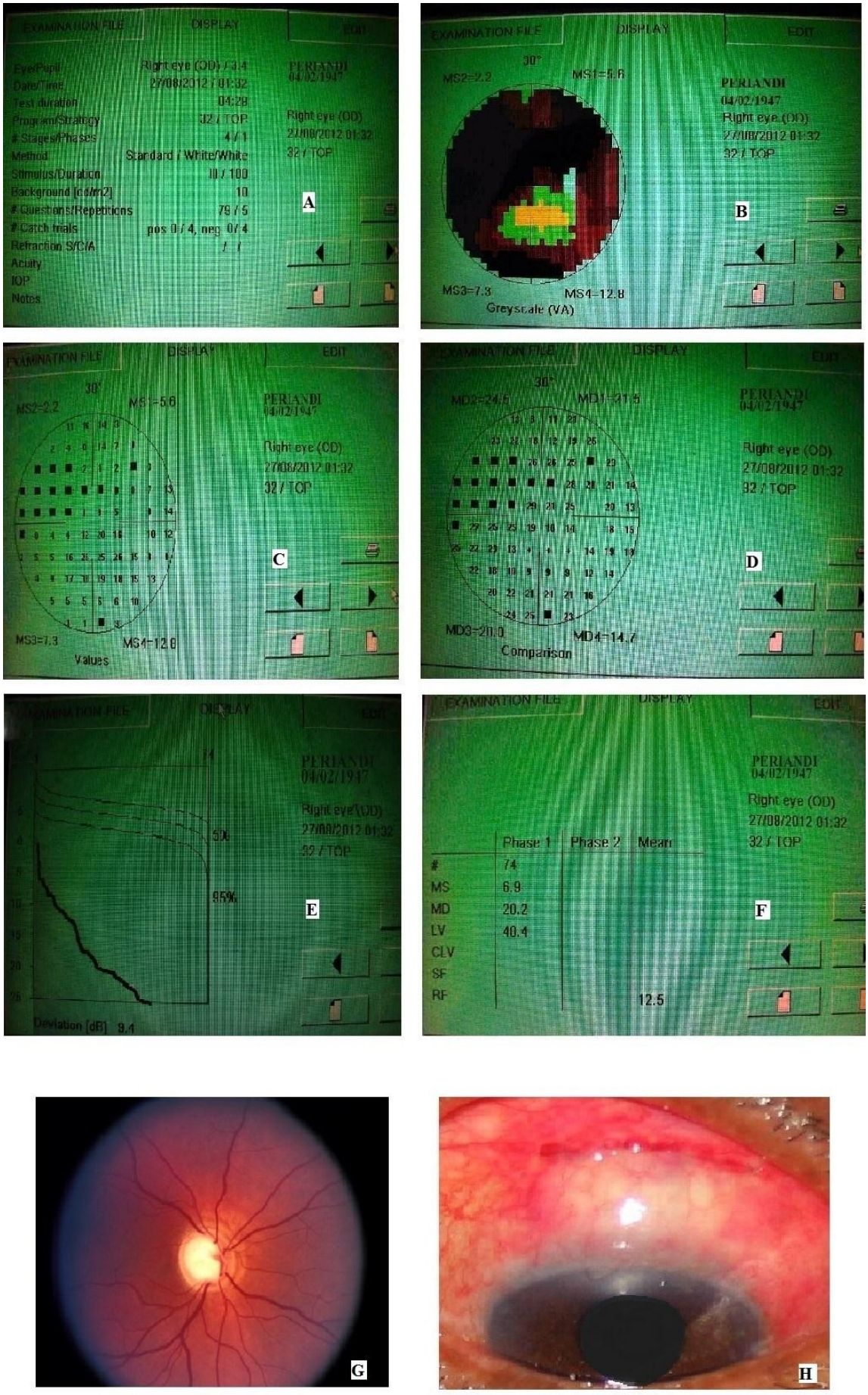


Fig.10. Glaucoma record of a study patient

A-F : Visual field right eye (Octopus) 32- TOP showing temporal island of vision

G: Fundus photograph right eye showing 0.9 glaucomatous cupping.

H: Clinical photograph right eye showing filtering bleb.

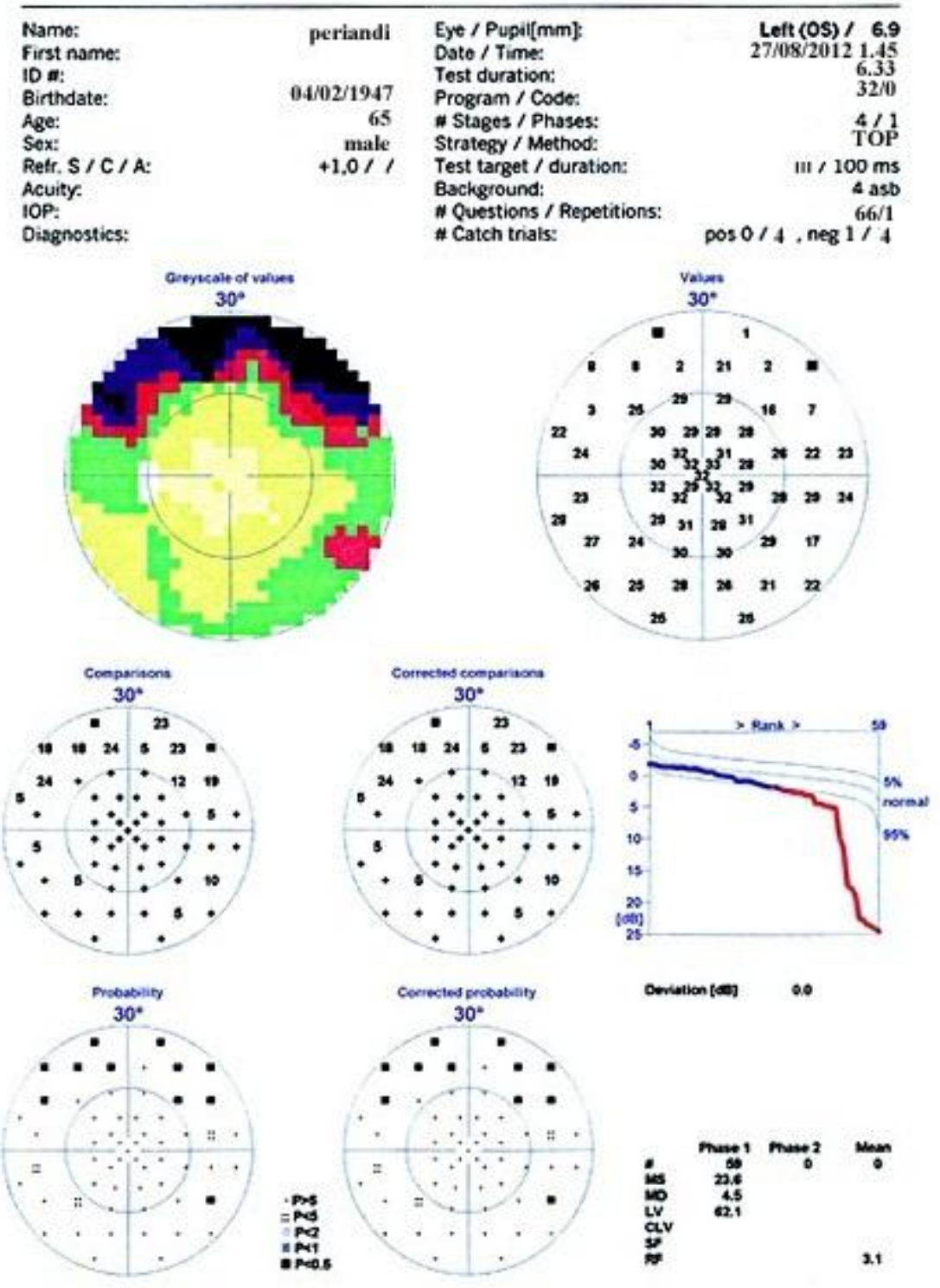


Fig.11 visual field left eye of a study patient.

Automated perimetry(Octopus) 32- TOP showing superior acuate scotoma

DISCUSSION

In our study, 56% were male and 44% were female. 7.1% of the males and 2.2% of the females were diagnosed to have primary open angle glaucoma. The high prevalence rate in males was comparable to other studies.

The overall prevalence of primary open angle glaucoma was 5% which is higher when compared to the South Indian population based prevalence study . The reason might be that our study was hospital based. A hospital based study by Smita et al in Northern India showed a high prevalence of POAG (33%). The prevalence of glaucoma suspect was 1%.

Defective vision was the common presenting complaint in our study which is similar to the study by Parikshit et al.[70] Among the primary open angle glaucoma patients, the visual acuity of two was significantly affected by cataract. Two had presenting visual acuity $\geq$6/24 and one had decreased visual acuity due to posterior capsular opacification.

Two of the POAG patients were already diagnosed to have glaucoma. Both had severe glaucomatous damage. They were using antiglaucoma medications in one eye and combined surgery had been done in the other eye. Inspite of this, one had increased IOP. She was followed up. CDR and field progression was noted in the eye with medical management. So she underwent combined surgery in the other eye also. The other post

trabeculectomy patient did not show any progression during the follow up period. He was continued on medical management in the other eye. Rest of the three POAG patients were started on medical treatment. They did not show any progression during follow up and had their IOP under control.

60% of the persons with POAG had not had the disease before our study. This is similar to the estimates found in the Visual Impairment Project (60%), 51% in Barbados Eye Studies and Blue Mountain Eye Study. In contrast to this, the rate of undiagnosed in APEDS was 92.6%, ACES was 93% and CGS was 98.5%.

20% of the newly detected patients had severe glaucomatous damage because patients above 40 years of age do not have routine eye check up to rule out the possibility of disease in them.

In our study, none of the patients had family history of glaucoma. Since only few of those with glaucoma are diagnosed and most of them are unaware about glaucoma, a family history of no glaucoma may be inaccurate. In Rotterrdam study, the first degree relatives of the patients diagnosed to have POAG were examined and 22.4% of them were found to have POAG.

In our study, 11.1% of the diabetics and 2.7% of the non- diabetics were found to have POAG. Our study did not show any significant association between diabetes and primary open angle glaucoma. The

percentage of POAG in diabetics is in excess compared to the VES (4.1%) and CGS (0%). 22% of the diabetics had diabetic retinopathy.

9.5%. of the POAG patients were hypertensives. Our study did not show any significant association between hypertension and POAG. This is similar to CGS. A hospital based study by Mohammed et al showed positive correlation between POAG and systemic hypertension.

IOP found with GAT was comparable with NCT in the right eye but there was a significant difference between them in the left eye. There was a significant difference between GAT and Schiotz in both left and right eye. However, GAT is considered as the gold standard.[71,72]

The drawback of our study was that IOP was not corrected for central corneal thickness due to the lack of pachymeter in our hospital.

In our study, 60% of the patients with POAG presented with an IOP >21 mm Hg. This is in contrast to the CGS in which only 32.81% presented with increased IOP. The diagnosis of glaucoma cannot be based only on increased IOP but increased IOP is an important risk factor.

The mean CDR in the right eye was 0.31 in non glaucomatous patients and 0.64 in the POAG patients. The mean CDR in the left eye was 0.32 in non glaucomatous patients and 0.62 in the POAG patients. There was a significant difference between the CDR of glaucomatous and non

glaucomatous patients in both eyes. The mean CDR in VES was 0.56 among the unselected population group and 0.39 in CGS.

There was a significant association between peripapillary atrophy and POAG in our study. 6% had peripapillary atrophy, of which 4% had POAG. Even though various differential diagnosis can be thought of for peripapillary atrophy, it is of prime importance to rule out POAG. The study by Kasner et al reported that the absence of peripapillary atrophy is associated with decreased risk of glaucomatous damage in ocular hypertensives.[73]

In our study, 80% of the primary open angle glaucoma patients had their visual field defect corresponding to the optic disc changes. There was a significant correlation between visual field defect and CDR.

In our study, 8% of the patients were aware of glaucoma. The other studies had varying figures. In spite of combined surgery been done in one patient, he was not aware of his disease.

In our study, the awareness of glaucoma was comparably low among both literates and illiterates. 14% of the literates and 4.1% of the illiterates were aware of glaucoma. It was statistically insignificant. This is in contrast to CGS and the study by Krishnaiah et al, in which the awareness increased exponentially with literacy. Hence education programs targeting the disease and its blinding potential must be increased. The aim of education should

focus on the benefits of early detection and treatment and changing the individual's perception about glaucoma.

Glaucoma awareness among the glaucoma diagnosed patients was also low.

In our study, 71% had previous eye check up. Among them, 4.2% had glaucoma. 33.3% of newly detected primary open angle glaucoma had previous eye check up when it was probably missed. The patients who have had previous eye check, glaucoma was not diagnosed. Even though the previous eye examination was done by the ophthalmologist, they were not detected to have glaucoma prior to our study. This is similar to ACES and CGS. An important risk factor for subsequent blindness is late diagnosis of glaucoma.

In our study, 29% of the patients did not have their eyes checked up in any form, by doctor, optometrist or nurse.

Unless optic disc evaluation is done as a routine, we would continue to have poor detection rates. According to Thomas et al,[74] poor examination techniques could also be related to primary training. Government eye camps should be conducted particularly for screening fundus to rule out glaucoma.

The referral sources are an important factor for early diagnosis. Other eye care personnel (ophthalmic assistants and optometrists) also must contribute.

The knowledge about glaucoma was 0%.

Glaucoma causes irreversible blindness and the affected people are unaware of their condition.In our study ,none of them were aware that the vision loss due to glaucoma was permanent and irreversible. Hence knowledge of glaucoma as a cause of irreversible blindness is 0%.

CONCLUSION

1.The overall prevalence of POAG was 5% in our study and the prevalence of glaucoma suspect was 1%.

2.Males had higher prevalence of glaucoma.

3.Diabetes and hypertension did not have any significant etiological association with POAG.

4.Intraocular pressure was not a definitive risk factor for screening, diagnosis and treatment of the patients.

5.Visual field changes had good correlation with the optic disc changes.

6.CDR and peripapillary atrophy had good correlation with POAG. Hence fundus examination is mandatory in all patients above 40 years of age.

7.60% of the glaucoma patients were newly diagnosed, of which 20% had severe glaucomatous damage.

8.Glaucoma awareness was extremely low considering the magnitude of blindness due to glaucoma.

9.There was no significant association between literacy and glaucoma awareness.

10.Previous eye check up and eye camps were not effective in creating awareness in the patients.

11.The knowledge about glaucoma was 0%.

12.In accordance with World Glaucoma Association, World Glaucoma Patient Association and Glaucoma Society of India, public, patients and the health care personnel should be tuned in both creating awareness and diagnosing glaucoma.

13.The family members of the glaucoma patients must be examined to rule out glaucoma.

14.Ophthalmoscopes should be provided to all ophthalmic assistants. They must be trained to look for glaucomatous optic disc cupping and report all suspicious optic discs. This would take glaucoma eye care to the rural areas.

15.Ophthalmologists must be trained to give special attention to glaucoma.

16.All eye care personnel should counsel the patients about the irreversible blindness caused by glaucoma.

17.It is high time media based health education focuses on glaucoma.

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PROFORMA

Name : **Age:** **Sex:**

Op No: **Occupation:** **Socioeconomic status :**

Address: **Phone number :**

History:

O Defective vision O Headache O Defective night vision

O Field loss O Coloured halos `O Frequent change of glasses

Past History :

O Diabetes O Hypertension O Myopia O COPD / asthma

O Trauma O Use of steroid eye drops O Acute fluid loss

O Blood loss during surgery/trauma O Major surgery

O H/O refractive surgery(excimer laser) O Shock during anaesthesia

O Raynaud's phenomenon/vasospastic episodes O Migraine

O Self antihypertensives intake without prescription

Family History :

Known glaucoma patient - First degree relatives/others

Personal History :

O Smoking O Alcohol consumption

EXAMINATION :

Media status : OD : OS :

Distant vision : OD : OS :

Near vision : OD : OS :

Refraction : AR : OD : OS :

Sub: OD : OS :

IOP : OD OS

Schiotz

GAT

NCT

Fundus :

1) Disc - overall size/ cup/ CDR(vertical)/ Asymmetry

2) Neuro retinal rim –colour/ thinning/notching

acquired optic pit/absence/translucency

3) Splinter haemorrhages

4) Peripapillary atrophy

5) RNFL(red free filter)

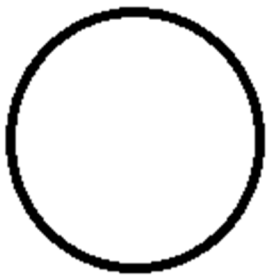
6) Vessels – Outpassing/Nasalization of vessels

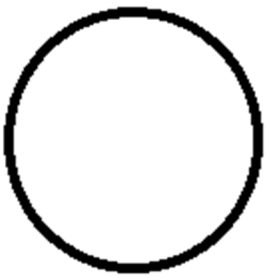
Baring of circumlinear vessels/Bayonetting of vessels

7) Laminar dots

8) Others

OD OS





Visual field analysis: OD OS

Overall sensitivity depression

Paracentral scotoma

Nasal step

Arcuate scotoma

Ring scotoma

Tubular vision

Temporal island

Others

Gonioscopy:

OD OS

Diagnosis :

GLAUCOMA AWARENESS QUESTIONNAIRE

Name Age Sex

Urban / Rural Literacy Socioeconomic Status

1) a) Have you ever heard of glaucoma

b) How did you come to know about glaucoma

c) Have you seen/heard/read any advertisement or publicity recently about glaucoma

2) a) What is glaucoma

- increase in pressure

- damage to nerve due to increased pressure in the eye

b) Symptoms of glaucoma

- defective vision

- pain /watering/redness

- other symptoms with good vision

c) Risk factors for glaucoma

Increased IOP, steroids, smoking, alcohol, family history of glaucoma, diabetes ,hypertension, migraine, myopia/ hypermetropia

d) Is glaucoma treatable

Therapies for glaucoma – eye drops , laser, surgery

Purpose of treatment

- irreversible on Rx

-permanent damage

-prevent progression

3) a) When is the last time you had eye check up

b) What is the earliest age you eye check up

c) Have you ever checked IOP / field

4) a) Has the eye doctor who checked you told about glaucoma

b) Did the physician tell you about glaucoma

5) a) Have you attended any eye camp

b) Has anybody told about glaucoma in the eye camp

6) Do you know that glaucomatous damage is irreversible

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு : திருநெல்வேலி மருத்துவக் கல்லூரி மருத்துவமனை, கண்பகுதியில் புறநோயாளியாக வரும் 40 அல்லது அதற்கு மேற்பட்ட வயதினரைப் பரிசோதித்து கண்அழுத்த நோயிருப்பைக் கண்டறிதல்.

(ஒளிவிளக்கு, கீற்று விளக்கு, உணர்வு நீக்கும் சொட்டு மருந்து ஊற்றி அழுத்தமானிகளால் கண்அழுத்தம் கண்டறிதல்; முன்னறைக் கோணத்தைக் கருவியால் பரிசோதித்தல், பார்வைக்களம் கண்டறிதல் முதலியன)

பங்கு பெறுவரின் பெயர் : வயது :

		பங்கு பெறுவர் இதனை 3 குறிக்கவும்
1	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை நான் படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2	நான் இவ்வாயில் தன்னிச்சையான தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4	இந்த ஆய்வில் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்கிறேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

ஆய்வாளரின் கையொப்பம் / ..... இடம் .....

ஆய்வாளரின் பெயர் .....

மையம் .....

கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை

சாட்சியின் கையொப்பம் / ..... இடம் .....

பெயர் மற்றும் விலாசம் .....

ABBREVATIONS USED IN THE MASTER CHART

M	Male
F	Female
CAD	Coronary artery disease
CVA	Cerebral vascular accident
COPD	Chronic obstructive pulmonary disease
BCVA	Best corrected visual acuity
G	Visual acuity $\geq$ 6/12
Mo	Visual acuity 6/18 – 6/60
P	Visual acuity < 6/60
PPA	Peripapillary atrophy
RE	Right eye
LE	Left eye
BE	Both eyes
NCT	Non contact tonometer
GAT	Goldmann applanation tonometer
POAG	Primary open angle glaucoma
NPDR	Non proliferative diabetic retinopathy
PDR	Proliferative diabetic retinopathy
HTR	Hypertensive retinopathy
POST TRAB	Post trabeculectomy

S.NO	NAME	AGE	SEX	DEFECTIVE VISION	DIABETES	HYPERTENSION	COPD	OTHERS	FAMILY HISTORY	CATARACT	BCVA		RE(mm Hg)			LE(mm Hg)			CUP/DISC		PPA		VESSEL CHANGES		FIELD CHANGES		GONIOSCOPY	DIAGNOSIS	HEARD OF GLAUCOMA	PREVIOUS EYE CHECKUP	ANYBODY TOLD ABOUT GLAUCOMA IN PREVIOUS EYE CHECKUP	BLINDING DISEASE
											RE	LE	SHIOTZ	NCT	GAT	SHIOTZ	NCT	GAT	RE	LE	RE	LE	RE	LE	RE	LE						
1	BALA	55	M	-	-	-	-	-	-	-	G	G	17.3	14	16	17.3	12	12	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
2	HELINA	42	F	-	-	-	-	-	-	-	G	G	14.6	14	14	14.6	17	14	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
3	VELAMMAL	60	F	+	-	-	-	-	-	-	P	G	14.6	12	14	14.6	12	14	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
4	SUBALAKSHMI	45	F	+	+	-	-	-	-	+	Mo	Mo	14.6	13	16	14.6	15	16	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
5	VALITHANGAM	60	F	+	+	+	-	-	-	+	Mo	Mo	22.4	23	22	12.2	13	12	0.6	0.9	-	+	+	+	+	+	IV	BE POAG /LE POST TRAB	+	+	+	-
6	DAISY	65	F	+	-	+	-	-	-	-	G	Mo	13.4	19	18	14.6	19	18	0.4	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	+	+	-	-
7	RAMASAMY	70	M	+	-	-	-	CAD	-	+	P	Mo	14.6	14	12	14.6	14	12	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
8	SHIVARAMAN	65	M	+	+	-	-	-	-	+	Mo	Mo	17.3	19	20	17.3	19	20	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
9	MUTHUMALAI	65	F	+	+	-	-	-	-	+	Mo	Mo	17.3	15	16	17.3	15	16	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA /BE MILD NPDR	-	+	-	-
10	PAPPAMMAL	56	F	+	-		-	-	-	+	P	P	12.2	9	10	12.2	8	10	0.4	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
11	KARUTHAIAH	65	M	-	-	-	-	-	-	-	Mo	G	20.6	20	18	20.6	20	18	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
12	ANNAPUSHPAM	47	F	+	-	-	-	-	-	-	P	P	14.6	11	10	14.6	11	12	0.4	0.4	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
13	SORIMUTHU	53	M	+	-	-	-	-	-	+	P	P	14.6	8	14	14.6	9	14	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
14	VALLI	43	F	-	-	-	-	-	-	-	G	G	17.3	15	16	17.3	14	14	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
15	KRISHNAMMAL	60	F	+	+	-	-	-	-	-	Mo	Mo	12.2	10	10	14.6	12	12	0.4	0.4	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-

16	ABDUL RAHMAN	48	M	+	+	+	-	CAD	-	-	Mo	Mo	22.4	25	22	24.4	22	22	0.4	0.4	-	-	+	-	+	-	IV	RE POAG	-	-	-	-
17	NAMBIRAJAN	71	M	+	+	-	-	-	-	+	Mo	Mo	10.2	10	10	10.2	11	10	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
18	KANTHAN	67	M	+	-	+	-	-	-	+	P	P	15.9	17	16	20.6	20	18	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
19	CHINNAMMAL	50	F	-	-	-	-	-	-	-	G	G	17.3	15	16	20.6	17	18	0.2	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
20	MUTHAIAH	82	M	-	-	-	-	-	-	-	G	G	10.2	11	9	10.2	11	10	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
21	SENDU	70	F	-	-	-	-	-	-	-	G	G	20.6	18	18	17.3	16	18	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
22	KRISHNASAMY	59	M	+	-	-	-	-	-	+	G	Mo	17.3	15	16	17.3	14	16	0.4	0.4	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
23	SHANMUGAVEL	52	M	+	+	-	-	-	-	+	P	P	14.6	12	10	14.6	11	10	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
24	JANBEGAM	53	F	-	-	-	-	-	-	-	G	G	17.3	16	16	17.3	15	14	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
25	PERIAMANI	70	F	+	-	-	-	-	-	+	P	Mo	12.2	10	12	12.2	8	10	0.4	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
26	RANJITHAM	60	F	+	-	-	-	-	-	+	P	P	20.6	14	18	17.3	15	16	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
27	PARVATHY	55	F	+	-	-	-	-	-	+	Mo	Mo	15.9	12	16	17.3	16	18	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
28	ARUNACHALAM	63	M	+	-	+	-	-	-	+	G	P	12.2	10	8	12.2	10	8	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
29	FATIMA	60	F	+	-	-	-	-	-	+	G	P	20.6	16	18	17.3	15	16	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
30	PHILOMINAL	65	F	-	-	-	-	-	-	-	G	G	14.6	14	12	14.6	13	12	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	+	+	+	-
31	VENKATRAJ	64	M	+	-	-	-	CVA	-	+	P	P	17.3	16	18	17.3	15	16	0.4	0.3	+	+	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
32	SUBBIAH	50	M	+	-	-	-	-	-	+	P	P	14.6	13	12	14.6	12	10	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
33	SARASWATHI	65	F	+	-	-	-	-	-	+	P	P	12.2	14	12	12.2	15	12	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
34	ANNAKILI	55	F	+	-	-	-	-	-	+	Mo	P	14.6	16	14	14.6	16	14	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
35	PERUMAL	70	M	+	-	-	-	-	-	-	G	P	12.2	15	12	14.6	16	12	0.3	-	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
36	THANGARAJ	45	M	+	-	-	-	-	-	+	Mo	Mo	10.2	9	8	8.5	9	8	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
37	REVATHI	43	F	+	-	-	-	-	-	+	P	P	14.6	15	14	14.6	15	14	0.1	0.1	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
38	SARASWATHI	54	F	-	-	-	-	-	-	-	G	G	10.2	11	10	10.2	13	11	0.3	0.4	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
39	ANANDARAJ	70	M	+	+	-	-	-	-	+	Mo	P	12.2	10	12	12.2	11	12	0.4	-	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
40	CHELLADURAI	53	M	+	-	+	-	-	-	+	Mo	Mo	14.6	13	12	14.6	16	14	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
41	KANNAMAL	65	F	+	-	-	-	-	-	+	P	P	17.3	15	14	17.3	15	14	-	-	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
42	DHANUKODI	60	M	-	-	+	-	-	-	-	Mo	Mo	14.6	15	12	14.6	14	12	0.3	0.3	+	+	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
43	PERIANDI	65	M	+	-	-	-	-	-	-	P	G	12.2	12	10	12.2	12	11	0.9	0.5	+	+	+	+	+	+	IV	BE POAG /RE POST TRAB	-	+	-	-
44	MARIAMMAL	60	F	-	-	-	-	-	-	-	G	Mo	10.2	11	10	10.2	13	11	0.4	0.4	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
45	FLORANCE	50	F	-	-	-	-	-	-	-	G	G	14.6	15	18	14.6	17	16	0.4	0.4	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-

46	AYYASAMY	70	M	-	-	-	-	-	-	+	P	P	17.3	13	14	12.2	12	10	-	-	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
47	JAYASEELI	62	F	-	-	+	-	-	-	-	Mo	Mo	14.6	11	10	14.6	11	12	0.3	0.2	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
48	THANGAIAH	56	M	+	+	-	-	-	-	+	P	P	12.2	11	8	12.2	11	10	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
49	MAGAMAYI	65	F	+	-	+	-	-	-	+	Mo	Mo	14.6	15	12	14.6	15	12	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
50	AKBAR	65	M	-	-	-	-	-	-	+	Mo	Mo	14.6	14	12	14.6	13	12	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
51	MARIAPPAN	66	M	+	+	-	-	-	-	+	Mo	Mo	17.3	19	18	17.3	17	18	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
52	SASTAKUTTY	70	M	-	-	-	-	-	-	+	Mo	Mo	17.3	18	18	12.2	10	8	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
53	MUTHU	53	M	-	+	-	-	-	-	-	G	G	12.2	14	14	12.2	10	14	0.4	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA /BE MILD NPDR	+	+	+	-
54	DAVID	45	M	-	-	-	-	-	-	-	G	G	10.2	12	10	10.2	12	10	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
55	MOHAMMED	73	M	+	-	-	+	-	-	+	P	P	24.4	23	22	26.6	33	26	0.6	0.9	-	+	+	+	+	+	IV	BE POAG	-	+	-	-
56	SUBBIAH	76	M	+	+	+	-	-	-	+	P	P	20.6	20	18	20.6	20	18	0.3	0.4	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
57	PAULRAJ	67	M	-	-	-	+	-	-	-	Mo	Mo	14.6	13	16	14.6	15	14	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
58	ISAKIMUTHU	63	M	+	+	+	-	-	-	+	Mo	Mo	17.3	19	16	17.3	18	18	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
59	VARALAKSHMI	55	F	+	+	+	-	CAD	-	+	P	Mo	17.3	18	18	14.6	10	8	0.4	0.4	+	+	-	-	-	-	IV	NO GLAUCOMA /BE PDR	-	+	-	-
60	KARUTHAN	75	M	-	-	-	-	-	-	-	Mo	P	15.9	14	12	15.9	16	12	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
61	KANNIAMMAL	42	F	+	-	-	-	-	-	+	P	Mo	20.6	20	18	20.6	20	18	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
62	KADAKARAI	64	M	+	+	+	-	-	-	+	Mo	P	14.6	14	14	14.6	16	14	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA /BE HTR	-	+	-	-
63	KARUPUSAMY	64	M	-	+	+	-	-	-	-	G	G	15	14	16	15	12	14	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
64	MARIAMMAL	45	F	+	-	-	-	CAD	-	+	P	G	13.4	13	12	13.4	16	14	-	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
65	UMA	51	F	+	-	-	-	-	-	+	Mo	Mo	20.6	18	16	20.6	17	18	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
66	SIVAGAMI	65	F	-	-	-	-	-	-	-	G	G	13.4	8	8	13.4	9	8	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
67	JOHNSON	52	M	+	-	+	-	-	-	+	Mo	P	13.4	12	12	13.4	14	12	0.2	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
68	EASWARI	61	M	-	+	+	-	-	-	-	G	G	17.3	18	16	17.3	17	16	0.3	0.2	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
69	NELLAIVADIVU	68	F	+	-	+	-	-	-	+	P	Mo	14.6	15	14	14.6	15	14	-	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
70	ABRAHAM	70	M	-	-	-	-	-	-	+	G	P	20.6	19	18	17.3	18	16	0.3	0.4	-	-	-	-	-	-	IV	NO GLAUCOMA	+	+	+	-
71	CHELLAMMAL	65	F	-	-	-	-	-	-	+	P	P	14.6	15	12	14.6	13	12	0.2	0.2	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
72	SHENBAGAM	57	F	-	+	-	-	-	-	-	G	G	20.6	16	18	20.6	17	18	0.4	0.4	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
73	VELU	66	M	-	+	-	-	-	-	-	G	G	20.6	16	18	20.6	19	18	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-

74	MOHAMED	50	M	+	-	-	-	-	-	+	P	Mo	17.3	17	16	17.3	17	16	-	0.2	-	-	-	-	-	-	IV	NO GLAUCOMA	+	+	+	-
75	PITCHAIA	60	M	-	-	-	+	-	-	-	G	G	12.2	10	12	12.2	11	12	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	+	+	-	-
76	MAHESHWARAN	49	M	-	-	-	-	-	-	-	G	G	15.9	14	16	13.4	12	12	0.4	0.4	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
77	CHELAPPA	69	M	-	+	-	-	-	-	-	G	G	12.2	14	12	12.2	13	12	0.4	0.4	-	-	-	-	-	-	IV	NO GLAUCOMA /BE MOD NPDR	-	+	-	-
78	LAKSHMI	46	F	+	-	-	-	-	-	-	Mo	Mo	13.4	15	12	13.4	16	14	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
79	CHANDRABOSE	50	M	-	-	-	+	-	-	-	G	G	17.3	16	16	17.3	15	16	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
80	POTTUKANI	60	F	-	-	-	-	-	-	-	Mo	G	14.6	13	14	17.3	18	16	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
81	SAMI	65	M	+	-	-	-	-	-	+	Mo	Mo	17.3	15	16	17.3	13	14	0.2	0.5	-	-	-	-	-	-	IV	GLAUCOMA SUSPECT	-	+	-	-
82	VELTHAI	67	F	+	-	-	-	-	-	+	P	P	17.3	14	16	17.3	16	16	-	-	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
83	ARUMUGAM	55	M	+	-	+	-	-	-	+	Mo	Mo	14.6	15	16	20.6	18	20	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
84	KARUVELAM	73	M	+	-	-	-	CKD	-	+	Mo	Mo	11.2	14	12	11.2	11	10	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
85	SHIVA	66	M	+	+	-	-	-	-	+	Mo	Mo	14.6	15	14	14.6	15	14	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA/ BE SEVERE NPDR	+	+	+	-
86	LAKSHMI	60	M	-	-	-	-	-	-	-	Mo	Mo	20.6	19	21	17.3	18	18	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
87	PUDUMAI	51	F	-	-	-	-	-	-	-	G	G	17.3	18	18	13.4	12	12	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
88	KANNAN	60	M	+	-	-	-	-	-	-	Mo	Mo	12.2	9	10	12.2	11	10	0.2	0.2	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
89	ISAKIAMMAL	50	F	+	+	+	-	-	-	+	P	P	12.2	11	10	13.4	12	12	0.3	-	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
90	PASUPATHY	56	M	-	+	+	-	-	-	-	G	G	17.3	18	16	17.3	16	18	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA /BE MILD NPDR	-	-	-	-
91	MARIAPPAN	75	M	+	-	-	-	-	-	+	Mo	G	20.6	20	18	20.6	20	18	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
92	PRABAKARAN	60	M	+	+	+	-	CAD	-	+	P	P	12.2	11	10	12.2	11	12	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
93	MARIMUTHU	70	M	+	-	-	-	-	-	+	Mo	Mo	12.2	9	12	12.2	11	10	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
94	KANTHAMMAL	65	F	+	-	-	+	-	-	+	G	P	14.6	15	14	14.6	15	14	0.3	-	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
95	PONNAMMAL	61	F	+	-	-	-	-	-	+	Mo	Mo	14.6	14	12	12.2	13	12	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
96	MUTHU	63	M	+	-	-	-	-	-	+	P	Mo	17.3	19	18	17.3	17	18	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
97	VELAYUDAM	46	M	-	+	-	-	-	-	-	G	G	19.6	13	14	14.6	14	14	0.7	0.4	+	+	+	-	+	-	IV	RE POAG	-	-	-	-
98	SETHALAKSHMI	59	F	+	+	-	-	-	-	+	Mo	P	12.2	13	12	14.6	15	12	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	-	-	-
99	SELVARAJ	58	M	+	-	-	-	-	-	+	Mo	P	14.6	15	14	13.4	13	14	0.3	-	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-
100	THAYAMMAL	55	F	+	-	-	-	-	-	+	Mo	G	10.2	11	10	11.2	13	10	0.3	0.3	-	-	-	-	-	-	IV	NO GLAUCOMA	-	+	-	-